VOL. 9 No. 2 APRIL, 1952

BRITISH JOURNAL OF INDUSTRIAL MEDICINE

EDITOR
RICHARD SCHILLING

EDITORIAL COMMITTEE

J. M. BARNES
SIR FREDERIC BARTLETT
THOMAS BEDFORD
G. R. CAMERON
T. A. LLOYD DAVIES
C. M. FLETCHER
M. W. GOLDBLATT
A. BRADFORD HILL
DONALD HUNTER
R. E. LANE
A. MEIKLEJOHN
L. G. NORMAN
H. ORMOND-CLARKE
J. R. SQUIRE
EDITOR, *British Medical Journal*

APPOINTED BY THE
BRITISH MEDICAL ASSOCIATION AND THE ASSOCIATION OF INDUSTRIAL MEDICAL OFFICERS

CONTENTS

	PAGE
History of Lung Diseases of Coal Miners in Great Britain: Part II, 1875–1920. ANDREW MEIKLEJOHN	93
Pneumoconiosis of Coal Miners in North East England with Special Reference to the Durham Coalfield. R. I. MCCALLUM	99
The Mechanism of Dispersion of Coal Particles in the Lungs of Miners. A. POLICARD	108
A Sampling Procedure for Measuring Industrial Dust Exposure. P. D. OLDHAM and S. A. ROACH	112
Dust Sampling and Lung Disease. C. N. DAVIES	120
The Incidence of Bladder Tumours in a Dyestuffs Factory. T. S. SCOTT	127
Urinary Coproporphyrins in Lead Poisoning. J. E. KENCH, R. E. LANE, and H. VARLEY	133
Studies on Cotton Dust in Relation to Byssinosis. Part I: Bacteria and Fungi in Cotton Dust. G. FURNESS and H. B. MAITLAND	138
Cardiovascular Disease in Cotton Workers. Part II: A Clinical Study with Special Reference to Hypertension. R. S. F. SCHILLING, N. GOODMAN, and J. G. O'SULLIVAN	146
Book Reviews	157
Abstracts	162

LONDON
BRITISH MEDICAL ASSOCIATION
TAVISTOCK SQUARE, W.C.1

YEARLY SUBSCRIPTION (4 NUMBERS) £2 2s. U.S.A $7.00 SINGLE NUMBER 12/6

GENERAL ADVISORY BOARD

A. J. Amor
Sir Henry Basford
G. P. Crowden
Sir Wilson Jameson
J. M. Mackintosh
E. R. A. Merewether
D. C. Norris
K. M. A. Perry
Donald Stewart
Sir Reginald Watson-Jones

President of the Association of Industrial Medical Officers and Hon. Editor of the Transactions of the Association of Industrial Medical Officers *ex officio*

APPOINTED BY THE
BRITISH MEDICAL ASSOCIATION AND THE ASSOCIATION OF INDUSTRIAL MEDICAL OFFICERS

NOTICE TO CONTRIBUTORS

The *British Journal of Industrial Medicine* is intended for the publication of original contributions in industrial medicine from workers of any nationality. It also provides sections for book reviews and abstracts.

All papers submitted for publication should be referred to Dr. Richard Schilling, Nuffield Department of Occupational Health, University of Manchester, Clinical Science Building, York Place, Manchester 13.

Papers are accepted on the understanding that they are contributed solely to this Journal, and that they are subject to editorial revision. Papers must be typewritten on one side of the paper only, with double spacing, and with a margin of at least 1½ in. Where half-tone reproduction of *x*-ray illustrations is required, authors should send in the original film and not prints. Photographs and photomicrographs should be printed on glossy paper, and should be unmarked. Charts and graphs accompanying papers should be carefully drawn in black ink on tracing linen or Bristol board or stout, smooth, white paper. Any lettering on these drawings to be done in the editorial office should be lightly inserted in pencil.

References should be arranged according to the Harvard system. When a book is referred to, the place and year of publication, edition and page should be given. In the text the year of publication must follow the author's name, more than one paper in any one year being indicated by a small letter (a, b, c) after the date. No numbering of references is necessary. At the end of the contribution references are arranged in the alphabetical order of the authors' names. The reference details are given as follows: Author's name, initials, year of publication (in parentheses), title of periodical (in italics, abbreviated according to the World List of Scientific Periodicals), volume number (bold type, Arabic numerals), and first page number (ordinary type, Arabic numerals), thus:

Dunn, C. W. (1940). *J. Amer. med. Ass.*, **115**, 2263.

Contributors will receive one proof in page, but it is assumed that all but verbal corrections have been made in the original manuscript; an allowance at the rate of ten shillings per sheet of sixteen pages is made for alterations in the proof (printer's errors excepted), and contributors will be responsible for any excess.

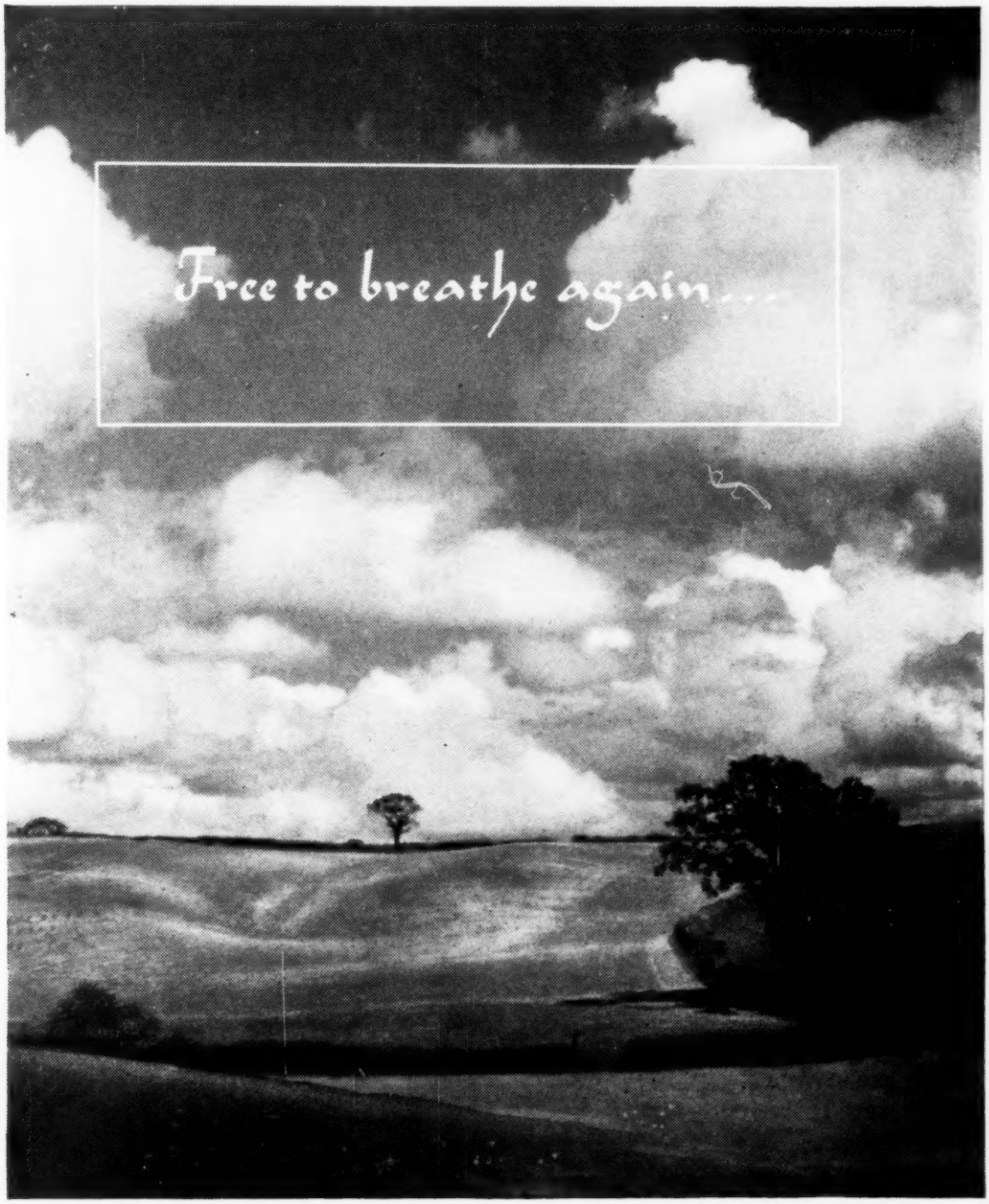
Twenty-five free reprints of articles will, if desired, be given to contributors. A limited number of additional reprints at cost price can be supplied if application is made when returning proofs. An estimate of costs will be given on application to the Publishing Manager, British Medical Association.

Papers which have been published become the property of the *British Journal of Industrial Medicine* and permission to republish must be obtained from the Editor.

Application for advertisement space should be addressed to the Advertisement Manager, British Medical Association, Tavistock Square, London, W.C.1.

NOTICE TO SUBSCRIBERS

Subscriptions are payable to the British Medical Association. Address: British Medical Association House, Tavistock Square, London, W.C.1.



FOR THE RELIEF of bronchial asthma, a choice of 'Neo-Epinine' preparations is available. Almost immediate relief is obtainable by oral inhalation of No. 1 Spray Solution, a plain 1 per cent aqueous preparation. The 20 mgm. compressed products, placed beneath the tongue, act within 5-10 minutes. Stubborn cases need oral inhalation of No. 2 Compound Spray Solution which contains 1 per cent of drug with 2 per cent of papaverine and 0·2 per cent of atropine methonitrate.

'Neo-Epinine' BRAND
Isoprenaline Sulphate
IN THE TREATMENT OF ASTHMA

BURROUGHS WELLCOME & CO. (The Wellcome Foundation Ltd.) LONDON

GENERAL ADVISORY BOARD

A. J. Amor
Sir Henry Bashford
G. P. Crowden
Sir Wilson Jameson
J. M. Mackintosh
E. R. A. Merewether
D. C. Norris
K. M. A. Perry
Donald Stewart
Sir Reginald Watson-Jones

President of the Association of Industrial Medical Officers and Hon. Editor of the Transactions of the Association of Industrial Medical Officers *ex officio*

APPOINTED BY THE
BRITISH MEDICAL ASSOCIATION AND THE ASSOCIATION OF INDUSTRIAL MEDICAL OFFICERS

NOTICE TO CONTRIBUTORS

The *British Journal of Industrial Medicine* is intended for the publication of original contributions in industrial medicine from workers of any nationality. It also provides sections for book reviews and abstracts.

All papers submitted for publication should be referred to Dr. Richard Schilling, Nuffield Department of Occupational Health, University of Manchester, Clinical Science Building, York Place, Manchester 13.

Papers are accepted on the understanding that they are contributed solely to this Journal, and that they are subject to editorial revision. Papers must be typewritten on one side of the paper only, with double spacing, and with a margin of at least 1½ in. Where half-tone reproduction of *x*-ray illustrations is required, authors should send in the original film and not prints. Photographs and photomicrographs should be printed on glossy paper, and should be unmarked. Charts and graphs accompanying papers should be carefully drawn in black ink on tracing linen or Bristol board or stout, smooth, white paper. Any lettering on these drawings to be done in the editorial office should be lightly inserted in pencil.

References should be arranged according to the Harvard system. When a book is referred to, the place and year of publication, edition and page should be given. In the text the year of publication must follow the author's name, more than one paper in any one year being indicated by a small letter (a, b, c) after the date. No numbering of references is necessary. At the end of the contribution references are arranged in the alphabetical order of the authors' names. The reference details are given as follows: Author's name, initials, year of publication (in parentheses), title of periodical (in italics, abbreviated according to the World List of Scientific Periodicals), volume number (bold type, Arabic numerals), and first page number (ordinary type, Arabic numerals), thus:

Dunn, C. W. (1940). *J. Amer. med. Ass.*, **115**, 2263.

Contributors will receive one proof in page, but it is assumed that all but verbal corrections have been made in the original manuscript; an allowance at the rate of ten shillings per sheet of sixteen pages is made for alterations in the proof (printer's errors excepted), and contributors will be responsible for any excess.

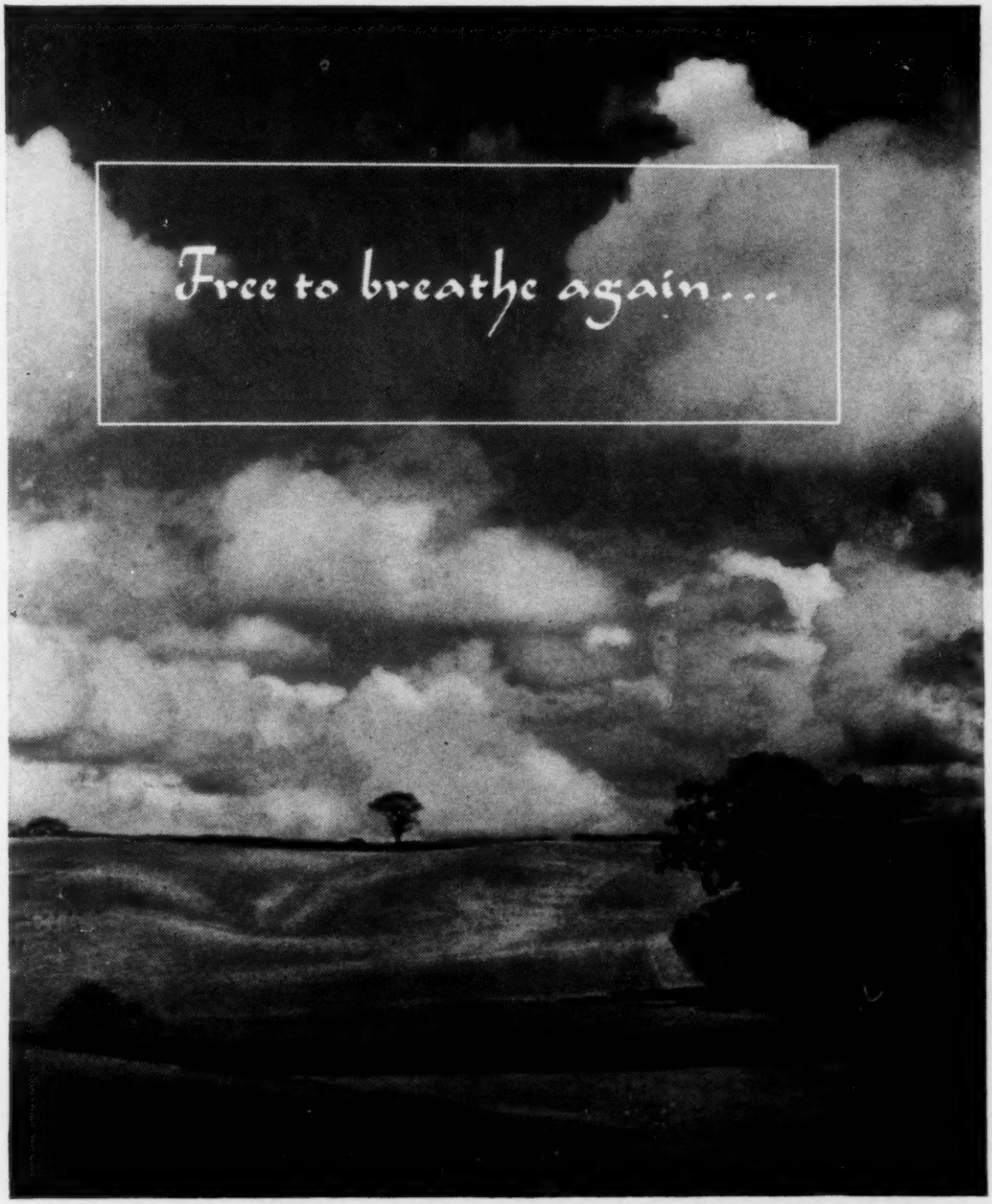
Twenty-five free reprints of articles will, if desired, be given to contributors. A limited number of additional reprints at cost price can be supplied if application is made when returning proofs. An estimate of costs will be given on application to the Publishing Manager, British Medical Association.

Papers which have been published become the property of the *British Journal of Industrial Medicine* and permission to republish must be obtained from the Editor.

Application for advertisement space should be addressed to the Advertisement Manager, British Medical Association, Tavistock Square, London, W.C.1.

NOTICE TO SUBSCRIBERS

Subscriptions are payable to the British Medical Association. Address: British Medical Association House, Tavistock Square, London, W.C.1.



FOR THE RELIEF of bronchial asthma, a choice of 'Neo-Epinine' preparations is available. Almost immediate relief is obtainable by oral inhalation of No. 1 Spray Solution, a plain 1 per cent aqueous preparation. The 20 mgm. compressed products, placed beneath the tongue, act within 5-10 minutes. Stubborn cases need oral inhalation of No. 2 Compound Spray Solution which contains 1 per cent of drug with 2 per cent of papaverine and 0·2 per cent of atropine methonitrate.

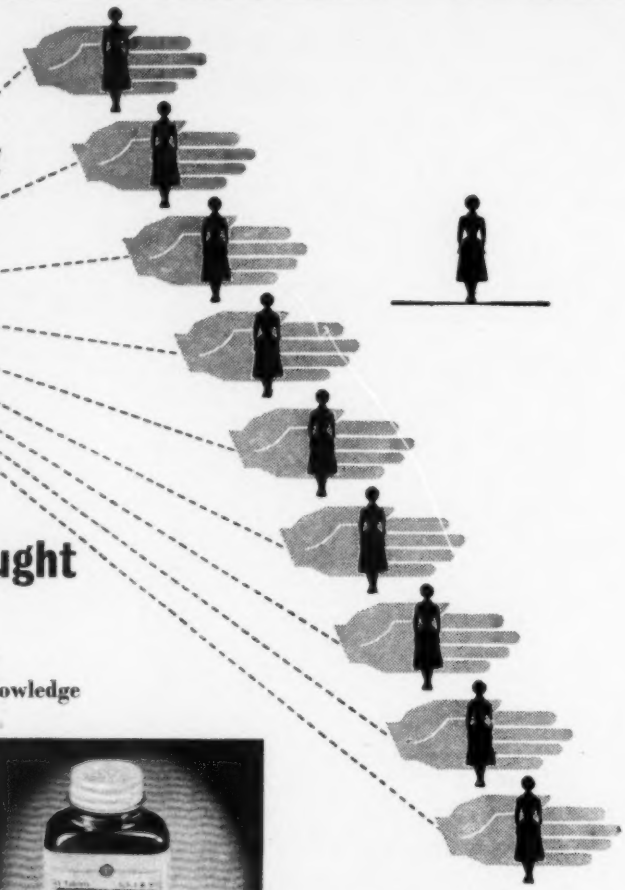
'Neo-Epinine' BRAND
Isoprenaline Sulphate
IN THE TREATMENT OF ASTHMA

BURROUGHS WELLCOME & CO. (The Wellcome Foundation Ltd.) LONDON

i

In dysmenorrhoea

9 out of every 10 received the relief they sought



'We use it [Edrisal] with the knowledge that 9 out of 10 sufferers will get the relief they seek, and that without loss of time they can return to the production line in complete comfort.'

Each tablet contains: amphetamine ('Benzedrine') sulphate 2·5 mg., acetylsalicylic acid 160 mg., phenacetin 160 mg.

Two studies* amongst factory and office workers revealed that with 'Edrisal' nine out of every ten dysmenorrhoea sufferers received the relief they sought. The analgesic properties of 'Edrisal' are enhanced by the unique anti-depressant properties of its 'Benzedrine' component. 'Edrisal', therefore, not only relieves the pain during the menstrual period but also combats the accompanying psychic depression. In primary dysmenorrhoea the best results are usually obtained with a dosage of two 'Edrisal' tablets, repeated every three hours, if necessary. Samples are available on request.

* *Industr. Med.*, **15**, 262, 1946; **15**, 679, 1946.

'EDRISAL'

Its dual action relieves pain . . . elevates mood

MENLEY & JAMES, LTD., 123 COLDHARBOUR LANE, LONDON, S.E.5

for *Smith Kline & French International Co., owner of the trade marks 'Edrisal' and 'Benzedrine'*

ESP41

Industrial Surgeries *now have the choice of two kinds of Elastoplast Plaster . . .*

FABRIC

PLASTIC

A fabric plaster made from lightweight cloth with lengthwise elasticity, and the new plastic type which stretches all ways and provides 100% waterproof protection when such is required. Both plasters provide adequate support for the injured part and have excellent adhesive properties. Full particulars or samples of either plaster will be sent, upon request, by the Medical Division of the Manufacturers.

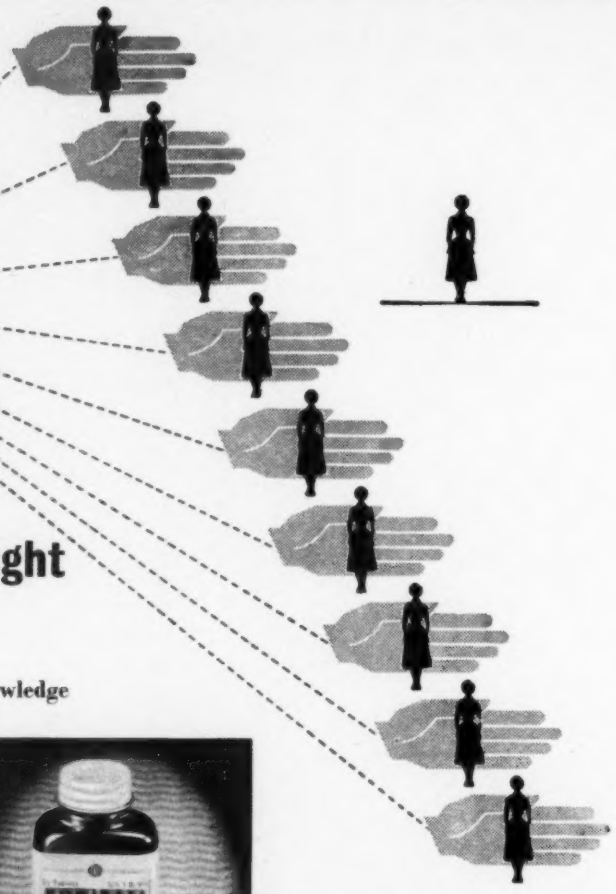
Elastoplast
TRADE MARK

Made in England BY T. J. SMITH & NEPHEW LTD., HULL
Outside the British Commonwealth, Elastoplast is known as Tensoplast

TWO KINDS OF PLASTER—CHOICE OF FABRIC OR WATERPROOF

In dysmenorrhoea

9 out of every 10 received the relief they sought



'We use it [Edrisal] with the knowledge that 9 out of 10 sufferers will get the relief they seek, and that without loss of time they can return to the production line in complete comfort.'

Each tablet contains: amphetamine ('Benzedrine') sulphate 2·5 mg., acetylsalicylic acid 160 mg., phenacetin 160 mg.

Two studies* amongst factory and office workers revealed that with 'Edrisal' nine out of every ten dysmenorrhoea sufferers received the relief they sought. The analgesic properties of 'Edrisal' are enhanced by the unique anti-depressant properties of its 'Benzedrine' component. 'Edrisal', therefore, not only relieves the pain during the menstrual period but also combats the accompanying psychic depression. In primary dysmenorrhoea the best results are usually obtained with a dosage of two 'Edrisal' tablets, repeated every three hours, if necessary. Samples are available on request.

* *Industr. Med.*, **15**, 262, 1946; **15**, 679, 1946.

'EDRISAL'

Its dual action relieves pain . . . elevates mood

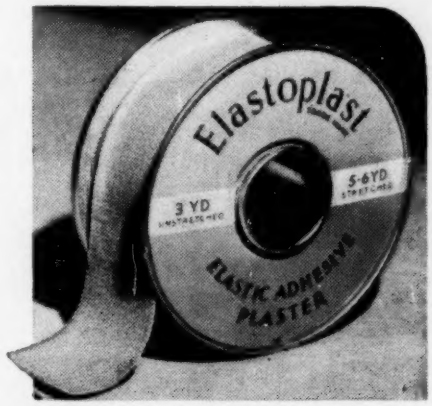
MENLEY & JAMES, LTD., 123 COLDHARBOUR LANE, LONDON, S.E.5

for *Smith Kline & French International Co., owner of the trade marks 'Edrisal' and 'Benzedrine'*

ESP41

Industrial Surgeries *now have the choice of two kinds of Elastoplast Plaster . . .*

FABRIC



PLASTIC

A fabric plaster made from lightweight cloth with lengthwise elasticity, and the new plastic type which stretches all ways and provides 100% waterproof protection when such is required. Both plasters provide adequate support for the injured part and have excellent adhesive properties. Full particulars or samples of either plaster will be sent, upon request, by the Medical Division of the Manufacturers.

Elastoplast
TRADE MARK

Made in England BY T. J. SMITH & NEPHEW LTD., HULL
Outside the British Commonwealth, Elastoplast is known as Tensoplast

TWO KINDS OF PLASTER—CHOICE OF FABRIC OR WATERPROOF

Save man-hours!

Time occupied
5 mins. per man

MOBILE X-RAY EXAMINATION ON SITE

For

INDUSTRIAL DISEASES OF THE CHEST

The Mobile Units of Portable X-Rays Ltd. X-ray employees on site at the factory, thus avoiding constant visits to hospital with the inevitable absence from productive work. The saving effected by this means will be found to be very considerable both in time and money.

The Mobile X-ray units of Portable X-Rays are now being regularly employed by many large industrial organisations. Demonstrations are offered free and without obligation.

Full details will gladly be sent on request.

**FREE DEMONSTRATION ON REQUEST*

PORTABLE X-RAYS

NIGHT Ltd & DAY

CHIswick 7836-7

POWER ROAD, LONDON, W.4.

CASELLA LONDON

AN

ELECTRO-MECHANICAL PARTICLE COUNTER

FOR

QUANTITATIVE MICROSCOPY

This instrument will enable existing work to be carried out both more rapidly and accurately. It will open up new fields of research throughout industry, medicine and biology, and will prove of particular interest to pathologists.

The counter, for which orders can now be accepted, is designed for straightforward counting, but within the next few months we also expect to have developed a counter which will automatically size-discriminate.

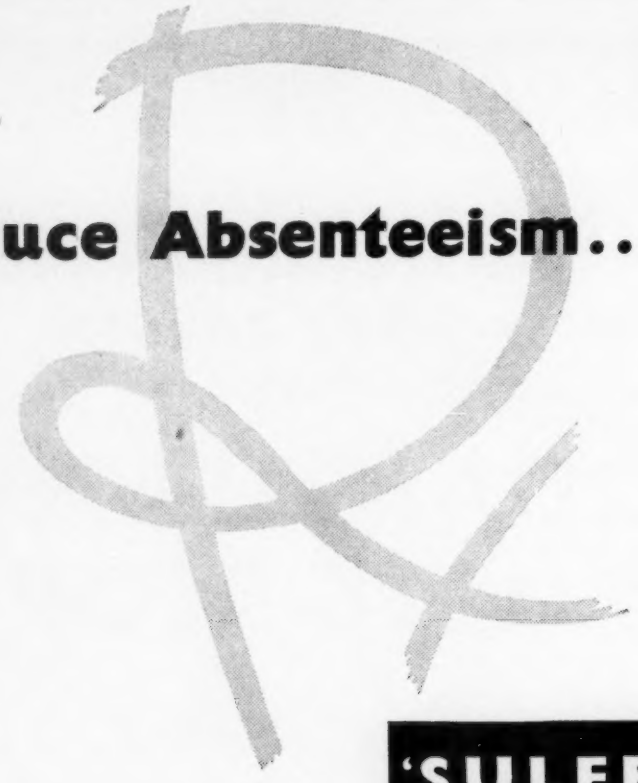
As the dimensions of the apparatus are only 48 × 20 × 18 inches it is suitable for use in any size laboratory.

C. F. CASELLA & CO. LTD.

REGENT HOUSE, FITZROY SQUARE, LONDON, W.1

Telephone : ★EUSton 3944

Member Scientific Instrument Manufacturers' Association of Great Britain



Reduce Absenteeism...

'SULFEX'

The works medical officer who prescribes 'Sulfex' does much towards reducing industrial absenteeism caused by the common cold. He provides not only relief from nasal congestion, but also prophylaxis against secondary invading organisms believed to be responsible for the severe late manifestations of the common cold. 'Sulfex' administered intranasally, at the first sign of a cold, is a valuable ally for promoting recovery.

'SULFEX' — Vasoconstriction in minutes...

...bacteriostasis for hours

MENLEY & JAMES, LIMITED, COLDHARBOUR LANE, LONDON, S.E.5
for *Smith Kline & French International Co., owner of the trade mark 'Sulfex'*

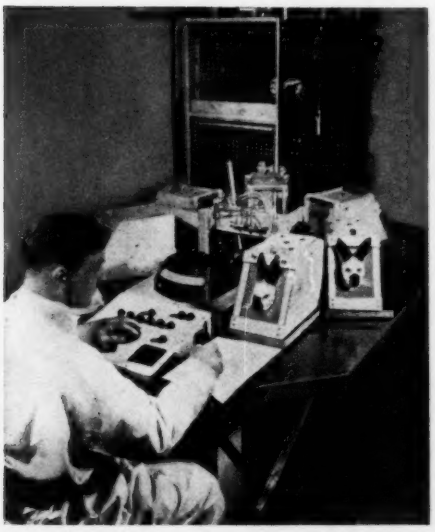
SXP10X

Intradex BRITISH DEXTRAN

A plasma substitute developed by British Scientists in Great Britain

The sixteen physical, chemical and biological tests, to which each batch of Intradex is subjected, ensure uniformity, sterility and freedom from toxic, pyrogenic, antigenic, anaphylactoid and cutaneous reactions.

It is interesting that each of a group of eight chinchilla rabbits, used to determine the renal excretion index, has received an average of approximately 1000 ml. of Intradex over a period of one year. This is equivalent to approximately 125 bottles (each 540 ml.) in a human subject. All these rabbits are in excellent condition.



Biological Control

The standard for freedom from pyrogenicity is more stringent than the B.P. standard. Rabbits are injected intravenously with 20 ml. Intradex per kg. of body weight. The batch of Intradex is rejected if any single rabbit in a group of six has a rise in temperature greater than 0.6°C. within four hours of the injection.

★

Available in M.R.C. transfusion bottles. Intradex (Salt Free) can be supplied for paediatric use and for the treatment of nephrotic cases. Further information and literature on request.

★

Intradex is the brand of dextran tested by the Medical Research Council and supplied to the Ministry of Health in Gt. Britain.

Manufactured by :

DEXTRAN LIMITED

AYCLIFFE · DARLINGTON

Sole Distributors :

THE CROOKES LABORATORIES LIMITED · PARK ROYAL · LONDON · N.W.10

When the problem is ***haemorrhage*** *or* ***shock***

'PLASMOSAN'
trade mark *brand*
polyvinylpyrrolidone solution

fulfils the requirements of a plasma substitute in that

1 Its viscosity, colloidal osmotic pressure and tonicity approximate closely to those of blood.

2 It has a high margin of safety, is non-antigenic and non-pyrogenic.

3 It is retained in the body for a sufficient time to allow the restoration of the plasma proteins.

4 It is not stored in the tissues.

5 It does not affect kidney function nor disturb blood grouping or clotting.

'PLASMOSAN' provides a sound and effective means of restoring the blood pressure in cases of injury, and of maintaining it during operation. Its particular value is that it is ready for immediate use in any amount, and can be administered without the time-consuming investigations which are necessary before blood transfusion.

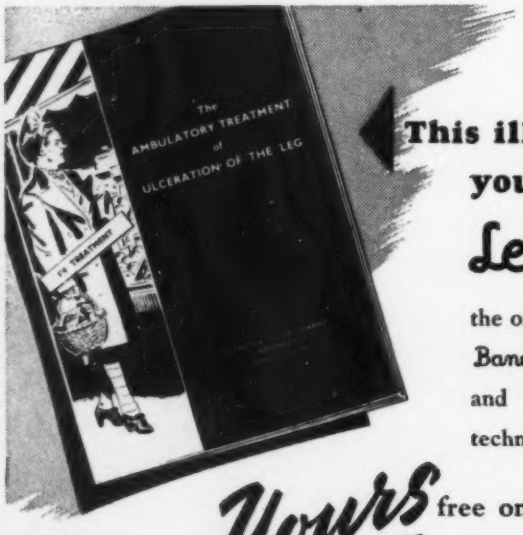
We shall be pleased to send detailed literature on request.

'PLASMOSAN' *is supplied in standard transfusion bottles of 540 c.c. in cartons of 4.*

manufactured by
MAY & BAKER LTD

MA277

distributors
PHARMACEUTICAL SPECIALITIES (MAY & BAKER) LTD DAGENHAM



This illustrated brochure tells you all about

Lestreflex

the original Leicester-made easy-flex adhesive
Bandage
and demonstrates some proved successful techniques in modern plaster therapy

Yours **free on request, and well worth getting**

DALMAS

Dalmas Ltd ★ Junior Street ★ Leicester Telephone 65261

Brit. J. industr. Med., 1952, **9**, 93.

HISTORY OF LUNG DISEASES OF COAL MINERS IN GREAT BRITAIN: *PART II, 1875-1920

BY

ANDREW MEIKLEJOHN

From the University of Glasgow

(RECEIVED FOR PUBLICATION JANUARY 28, 1952)

" The prevention of disease is fully as reliable a testimony of professional skill and scientific knowledge as its treatment ; whilst it is generally considered to constitute a surer evidence of the possession of that disinterested and noble philanthropy which should ever characterise the disciples of physic." W. I. Cox (1857)

In a lecture to the Edinburgh Medical Society, delivered in 1883, and later published under the title of " Germs, Dust and Disease ", Dr. Andrew Smart expressed the opinion that anthracosis or " miners' consumption " had but a doubtful, if any, existence. Thereafter, he continued his study of coal miners from different districts and communicated his results in a " Note on Anthracosis " read at the annual meeting of the British Medical Association at Cardiff in 1885. The observations which he then presented confirmed his previous conclusions, particularly the non-existence of " anthracosic disease " and that pulmonary consumption was of exceptionally rare occurrence among coal miners. He discusses his findings thus :

> " Looking to their longevity, high standard of health, and low death-rate, especially from pulmonary disease, one is led to suppose that there must be some special protective feature in coal-mining operations not shared in by the rest of the dusty trades. The preserving element may, after all, be the dust derived from the coal, which has hitherto been credited with the opposite effect.
>
> The antiseptic properties of carbon are generally admitted ; and I can aver that, not infrequently, I have examined miners, who, for over fifty years, have daily respired the coal-dust laden air of the mines, with no other effects than the perfectly harmless staining of the sputa, and, it may be, in some instances, of the pulmonary tissue."

Occupational Mortality Statistics

A more general view of medical thought on the disease about this period was presented by Dr. Ogle (1885) in the supplement to the forty-fifth annual report of the Registrar-General, which relates to England and Wales for the 10 years 1871–1880.

In dealing with the mortality statistics of miners, Ogle emphasized certain difficulties ; first, that death certificates seldom distinguished between coal and other miners, and secondly, that frequently there existed confusion between phthisis and other affections of the respiratory organs. The former difficulty was largely surmounted by separating the deaths according to geographical areas, for as it happens, coal and metalliferous mining seldom overlap to any serious extent in the same area ; thus Northumberland is mainly coal mining, N. Yorkshire, ironstone mining, and Cornwall, tin mining. Ogle submits :

> " If we exclude accidents, it will be found that the mortality of the coal miners only slightly exceeds that of the most healthy class of men, the agriculturists, that is to say, the farmers, the agricultural labourers, and the gardeners."

Dr. Greenhow in 1858 had recorded that " whilst lead and copper and tin mining are certainly dangerous to health, coal mining appears to be at least not ' unhealthy ' ".

These statements, it was realized, might not be wholly true, due to the fact that miners are a body of picked men and that any who become weakly must, by the heavy nature of the work, abandon it for a lighter occupation.

When all due allowance was made for possible inaccuracies, Ogle concluded :

> " It must be admitted that coal miners suffer from diseases of the respiratory organ to a greater extent than those engaged in most other industries."

He did, however, emphasize " the comparative

* Part I was published in 1951 (**8**, 127).

innocuity of coal dust, as compared with stone-dust or metallic dust ".

Reference is made to the general concurrence of evidence, English, Belgian, and German, that " for some reason or other the mortality of coal miners from phthisical disease is excessively low as compared with that of other workers".

In relation to this Dr. Hirt (1871) of Breslau is quoted :

> " It is in the highest degree probable that coal-dust possesses the property of hindering the development of tuberculosis, and of arresting its progress."

While admitting that this thesis might ultimately be sustained, Ogle felt that, for various reasons, the conclusion, so far, had not been established beyond all possibility of fair doubt.

The Close of the Nineteenth Century

Meanwhile considerable attention was being focused on occupational diseases of the lungs among other groups of workmen, such as fork and cutlery grinders, earthenware workers, stonemasons, and sandstone quarrymen. This means that doctors were aware of the occurrence of these diseases so that the diminishing prevalence of respiratory disease in coal miners must have been impressive to attract such frequent emphatic record.

Arlidge (1892) in his textbook on " The Diseases of Occupations " comprehensively reviewed the whole subject. His testimony is specially valuable, for he was one of the most distinguished Fellows of the Royal College of Physicians of London, and for many years was consulting physician to the North Staffordshire Infirmary, which served a very extensive coal-mining area. Moreover, lung diseases, because of their serious prevalence among the local pottery workers, were his particular study. He records :

> " There is a widespread belief at the present day that the serious lesions of the lungs associated with the calling of coal-getters belong to past history, or, at the most are very uncommon ; and no doubt can exist that, compared with the past, they are becoming rarer, thanks to the introduction of efficient ventilation, of shortened hours of labour, and of the increased attention given to the hygiene of mines."

The following passage is remarkable for its minute and comprehensive clinical and pathological observations.

> " It may be affirmed generally of the dyspnoea of old miners, that it is out of proportion to the extent of disabled lung. Its production is not of uniform origin. One cause is the infarction or stuffing of the lung tissue with the foreign matter ; another is the bronchial trouble, with its attendant plugging with mucus and the thickening of the lining membrane of the tubes ; a third is the greater or less extent of tissue in an emphysematous state ; a fourth, active at times, is blood short of red corpuscles ; a fifth cardiac valvular disease, secondary to the respiratory disturbances ; and a sixth to derangement of nervous supply, consequent, perhaps, on the enlarged and diseased bronchial glands at the root of the lungs and in contiguity with the pulmonary plexus. At all events, paroxysmal coughing and breathing is a usual accompaniment of pulmonary fibrosis."

Arlidge did not subscribe to the current view of " the conservative influence of coal-dust in warding off tubercular consumption " and he believed that large fibrotic masses could break down without the presence of tuberculosis.

> " Further, in fibrosis, there is not the early breaking down of the newly-formed morbid tissues witnessed in tubercular formation. Not but that softening does occur in fibroid degeneration ; still, when it does happen, it begins in the central portion of the mass, and is the result of starvation on account of gradual degeneration and obliteration of blood-vessels. Nevertheless, it may be granted that a softening portion of cirrhosed lung may afford a suitable nidus for tubercular matter originating elsewhere ; and that when this happens we may anticipate more serious havoc. This occurrence is well-established by clinical experience."

So we reach the end of the nineteenth century, and thus far, in tracing the history of the rise and decline of coal-miners' pneumoconiosis, we have relied mainly on the recorded experience and opinions of practising physicians. The supplement to the sixty-fifth annual report of the Registrar-General published in 1908 provided occupational mortality statistics which serve to test these observations. It is recognized that these figures are not absolute measures but are subject to several errors. In presenting the statistics Tatham (1908) emphasized particularly the fallacies which attach to death certificates for the purposes of dividing phthisis, tuberculosis, and other respiratory diseases and in distinguishing primary disease from terminal complications. Comparison is made of the triennial periods 1890–92 and 1900–02 and the conclusion reached is that " there has been a considerable decline of mortality from disease, not only among miners in the aggregate but also in each section (geographical) of the occupation " (Table 1). Besides, not only did the decline occur in the separate areas but it was reflected in all age groups. Table 2, summarized from the report, indicates the favourable trend of respiratory diseases.

The Opening of the Twentieth Century

Despite the decline the subject continued to attract comment. Dr. Trotter, Medical Officer of Health, Bedlingtonshire U.D.C., Northumberland, writing (1903) on the " so-called anthracosis and phthisis in coal miners " records that " probably largely owing to the great improvements in mine

TABLE 1

COMPARATIVE MORTALITY OF COAL MINERS (OCCUPIED ONLY) AGED 25 TO 65 YEARS IN SELECTED AREAS

		Mortality All Causes	Disease	Accidents
All occupied males	1890–92	1102	1038	64
	1900–02	925	867	58
All coal miners	1890–92	1068	905	163
	1900–02	846	723	123
Coal miners in Lancashire	1890–92	1236	1057	179
	1900–02	1006	875	131
Coal miners in Monmouthshire and South Wales	1890–92	1322	1041	281
	1900–02	951	782	169
Coal miners in Staffordshire	1890–92	1100	943	157
	1900–02	846	728	118
Coal miners in West Riding	1890–92	1051	920	131
	1900–02	783	684	99
Coal miners in Durham and Northumberland	1890–92	894	783	111
	1900–02	763	658	105
Coal miners in Derbyshire and Nottinghamshire	1890–92	841	737	104
	1900–02	675	595	80

TABLE 2

COMPARATIVE MORTALITY OF COAL MINERS (OCCUPIED ONLY) AGED 25 TO 65 YEARS FROM RESPIRATORY DISEASES

Ref. No.	Occupation		All Respiratory Diseases	Phthisis	Bronchitis	Pneumonia	Pleurisy	Other Diseases of Respiratory System
	Occupied males (England and Wales)	1890–92	469	214	101	122	8	24
		1900–02	340	175	53	87	6	19
83	Coal miner	1890–92	423	113	131	141	8	30
		1900–02	274	85	75	85	6	23
83a	Coal miner (Durham and Northumberland)	1890–92	291	109	58	85	10	29
		1900–02	205	84	41	54	6	20
83b	Coal miner (Lancashire)	1890–92	567	118	198	217	14	20
		1900–02	386	96	113	149	14	14
83c	Coal miner (West Riding)	1890–92	473	142	137	165	7	22
		1900–02	247	88	67	71	5	16
83d	Coal miner (Derbyshire and Notts.)	1890–92	263	80	87	77	1	18
		1900–02	186	64	49	52	5	16
83e	Coal miner (Staffs.)	1890–92	463	95	204	127	9	28
		1900–02	277	66	104	71	6	30
83f	Coal miner (Monmouthshire and South Wales)	1890–92	522	124	153	190	5	50
		1900–02	343	93	104	108	5	33

ventilation in this country, these conditions are very uncommon now". He makes this interesting comment :

> " This blackening or anthracosis is by no means necessarily due to a diseased condition of the lungs, nor does it seem to be a factor in the causation of a diseased condition of the lungs. Phthisis, either fibrous or tuberculous, as a disease among coal miners due to their occupation is very uncommon indeed ; in fact, as a disease phthisis is probably more uncommon in coal miners than in any other class of workmen in the country."

When tubercular phthisis did occur in any of these cases he was definitely of opinion that it was due to the inhalation of particles from the " post or sandstone strata and the dust from the shale and seggar clay." As all other influences predisposing to tubercular phthisis were present in the area, he believed that coal dust had a protective influence. His conception of anthracosis is interesting :

> " Anthracosis as a disease per se does not exist, but is really an epiphenomenon in diseases of the lungs in which this blackening is found."

This contribution was immediately followed by two letters to the editor. Hall (1903) emphasized that Trotter had said nothing new, because Smart, whose contributions have already been noted, had presented all these matters in his clinical lectures at Edinburgh Royal Infirmary in 1892. Purdy (1903), however, who had previously been in practice at Cambois and Ashington in Northumberland, wrote adding his testimony that " the average coal miner often works in the mine under more healthy conditions than those in which he lives at home".

Miners' phthisis was the subject of discussion in the section of pathology at the meeting of the British Medical Association in the summer of 1903. Oliver (1903) of Newcastle-on-Tyne submitted the paper, which is particularly interesting by reason of details of dusting experiments carried out by him on laboratory animals, guinea-pigs and rabbits. The dust was introduced by insufflation, and he was mainly concerned to observe the distribution of the dust and its transport from the alveoli to the lymphatics by large round phagocytes or macrophages. The origin of these cells was obscure ; they are described as " either leucocytes, alveolar epithelial cells, or connective tissue corpuscles ". From references cited it is clear that French pathologists (Claisse and Josué, 1897) had been conducting such experimental studies for several years. Oliver's main conclusions are :

> " 1. It is a disease of occupation, due to the irritating action of dust, which renders the individual liable to repeated catarrh, that it is non-tuberculous, not only in the early stages, but may even remain non-tuberculous throughout ; that when tubercle is present it is an accidental infection, which while hastening the end, does not exercise any very marked influence upon the pre-existing structural alterations caused by dust, unless it be a tendency towards disintegration.
>
> 2. That it is a local disease, purely personal to the individual, and never assuming hereditary characters.
>
> 3. That accepting this view of the pathology of the disease, miners' phthisis, as experience in this country has shown, may be largely prevented by improved ventilation, and by the adoption of means for the removal of dust or for the allaying of dust by water."

Workmen's Compensation and Occupational Diseases

At this point it is necessary temporarily to interrupt the chronological story and return to the year 1880, which marks the first workmen's compensation act. This was the Employers Liability Act, which provided a measure of compensation to workmen injured by accident arising out of and in the course of their employment. The liability was restricted to accidents, traumatic injury, but meanwhile doctors were directing attention to the increasing toll of sickness and death caused by such occupational diseases as silicosis, poisoning by lead, arsenic, phosphorus, mercury, manganese, and many other substances. The legislature were constantly urged to extend the scope of compensation payments to include occupational diseases, and in 1903 Lord James of Hereford, by an award in Court, established lead poisoning in pottery workers as the first compensatable occupational disease. This precedent, having been achieved, workers were encouraged to press for the inclusion, in like manner, of other diseases.

So in 1906 a Departmental Committee on Compensation for Industrial Diseases was appointed under the chairmanship of Herbert Samuel, Esq., to enquire into what diseases might rightly be regarded as occupational and included in the schedule to the Act. In the course of their investigations considerable expert evidence was submitted on a variety of diseases including fibroid phthisis. Several doctors and laymen from colliery areas gave evidence on the diseases of coal miners and thereby we are enabled to form some idea about the occurrence of respiratory diseases among coal miners throughout the country.

The witnesses were almost unanimous that serious fibroid phthisis had considerably diminished in incidence. Dr. Scott, a physician to Glasgow Royal Infirmary, represents the general opinion ;

> " It was very common among miners when I began practice . . . . now it is rarely seen, even in hospital."

Black lungs, however, were still encountered in coal miners, and Dr. Bell of Dunfermline expresses the common attitude as to causation ;

> " It is undoubtedly at any rate in the first instance due to the inhalation of coal dust and the impregnation of the lung tissue with coal dust ;

and,

> " I must say that coal dust is not of itself very injurious either to the lung or to an ordinary wound, it is carbon,

and,

> " There is nothing very injurious about the ordinary coal dust except that it irritates to a certain extent and causes bronchitis, but it does not cause injury the same as steel dust, for instance, or copper dust and so on ; there is none of the wasting."

Based on his experience of 3,000 necropsies at the Western Infirmary, Glasgow, Professor Robert Muir testified that black lungs were benign and that he had not met the fibroid phthisis here such as he had seen in his department in Edinburgh, where he had had a wide experience of masons' phthisis. Coal dust, unless mixed with rock dust, was, in his opinion, innocuous.

Having regard to the modern problem of alternative employment for coal miners disabled by respiratory disease, the following passage from the examination of Mr. P. J. O'Keefe, St. Helens, Lancs., is of interest.

> " Have you known men who have left the work at 35 and taken up other work ? No, they will not do that. You will not get a collier to leave his employment. There are so few branches of employment for them, only labourers' work which is badly paid, so they will not go into that ; and even if they go into chemical works they require to have some training for it."
>
> " Do you think a periodical examination of these workers every six months, with power to suspend men who are beginning to show signs of the disease from the work would be useful ? I think it would."
>
> " Except that there is a difficulty about their finding other work ? Yes, there is that to contend with."

Among the witnesses there was one dissentient opinion. Dr. A. Rowley Moody of Hanley, North Staffordshire, asserted that " respiratory disease in potters is decreasing but increasing enormously in coal miners ". In explanation he adduced the following reasons :

> " 1. recruits to the mines no longer from country people but from weakly town-dwellers ; 2. the elimination of small mines ; 3. mines becoming deeper and more extensive."

He then proceeded to indicate that " the difficulties in the way of ventilation are, of course, largely increased, and the mines, many of them have got extremely dry and dusty ".

In due course the Committee reported :

> " We are clearly of opinion that coal miners are not liable to fibroid phthisis, and although cases of anthracosis, using the term to mean cases in which the lung is charged with coal dust, are commonly met with, we cannot find that in any one that condition has proved to be a contributory cause of death."

The Role of Free Silica in Pneumoconiosis

In the following year Oliver (1908) published his classic textbook on diseases of occupation, in which field he was regarded as a leading authority.

He records that in 1878, 30 years before, when he became a physician to the Newcastle Infirmary, miners' phthisis was extremely prevalent, but " so great has been the change which has come over the North of England coal miners that I can safely say there has scarcely been a case of coal miners' phthisis in the wards during the last ten years ".

He attributes the disease to previous faulty conditions underground and the improvement to more efficient ventilation. On the occurrence of pulmonary consumption among coal miners, he is among those who support the view that " the occupation rather protects him from, than predisposes him to pulmonary tuberculosis ".

In 1914, in a series of five Milroy Lectures delivered before the Royal College of Physicians of London, Shufflebotham, of Stoke-on-Trent, North Staffordshire, devoted himself to a comprehensive review of the hazards of coal-mining in the United Kingdom. For him, however, the real importance of dust in coal mines was in relation to underground explosions. This is significant especially as he did comment on respiratory diseases.

> " Though fibroid phthisis does not affect coal miners at the present time in this country, still anthracosis, or infiltration of the lung with coal dust, is widely found ". . .

and again

> " Fibroid lung (such as potters suffer from) is rare among coal miners, and anthracosis does not seem to entail disablement. . . . At the present time in Great Britain fibrosis of the lung among miners can be said to be practically non-existent and its disappearance can be wholly attributed to the enforced improvements in the ventilation of mines, in illumination [this probably refers to greater use of safety lamps in place of naked lights], and to shorter working hours."

The next series of Milroy Lectures, delivered by E. L. Collis (1915), was devoted to industrial pneumoconioses. In the literature these have attained to the eminence of a medical classic. Of coal miners he records that " fifty years ago asthma was common among them ", and he later adds " the disease, however, common though it used to be, has passed almost unobserved from our midst, and conjectures as to its character and causation are idle." His researches revealed that bronchitis varied in its prevalence in different coalfields, thus it was common among the coal miners in Lancashire and Monmouth and South Wales, while in Northumberland, Durham, and Derbyshire it was practically unknown. This difference he explained by the fact that in the former areas there was considerable faulting and

the lung damage was induced by the rock or sandstone dust :

> "The mortality of Lancashire coal miners condemns dust of the strata between the coal seams. . . .
> "Coal dust then is not injurious, but because it can be easily distinguished, it is useful to demonstrate the path by which dust enters and travels through the lungs."

His final conclusion, which is, in fact, his main thesis, is important.

> "I have attempted to justify the claim that dust inhalation plays an important part in determining the occurrence of respiratory diseases, some dusts such as coal, it is true, not only appear to have no power of producing pneumoconiosis, but even may possess some inhibitory influence on phthisis ; but most dusts have an injurious influence, and of all dusts that of silica is most injurious."

Thereby the whole emphasis of noxious dust was placed on free crystalline silica and that particular variety of pneumoconiosis, silicosis. This conclusion almost entirely dominated the subject for the next 15 years, until 1930, by which year asbestosis, a fibroid disease of the lung due to combined silica, had been identified by Cooke (1924) and accepted by experts as of occupational origin.

In 1919 silicosis for the first time was recognized as a compensatable disease, but only under certain conditions and only in respect of workmen employed in the refractories industries, that is to say in men employed in processes carried on at mines, quarries, factories, and workshops, at which refractory material containing not less than 80% total silica (SiO_2) is got or manipulated with a view to manufacture or sale. This event may be regarded as the beginning of the present phase.

(To be concluded)

References

Arlidge, J. T. (1892). "The Hygiene, Diseases and Mortality of Occupations." Percival. London.

Claisse, P., and Josué, O. (1897). *Arch. Méd. exp.*, **9**, 205.

Collis, E. L. (1915). "Industrial Pneumonoconioses." (Milroy Lectures, 1915). H.M.S.O. London, 1919. (Reprinted from *Publ. Hlth. Lond.*, 1915, **28**, 252, 292 ; **29**, 11, 37.)

Cooke, W. E. (1924). *Brit. med. J.*, **2**, 147.

Departmental Committee on Compensation for Industrial Diseases (1907). Report. (Cd. 3495 and 3496.) H.M.S.O. London.

Greenhow, E. H. (1858). Papers relating to the sanitary state of the people of England. General Board of Health. H.M.S.O. London.

Hall, W. T. (1903). *Brit. med. J.*, **1**, 1410.

Hirt, L. (1871). "Die Krankheiten der Arbeiter, abt 1, t. 1 : Die Staubinhalations-Krankheiten. Breslau.

Ogle, W. (1885). Supplement to 45th Annual Report of Reg.-Gen. for England and Wales. Cd. 4564. H.M.S.O., London.

Oliver, T. (1903). *Brit. med. J.*, **2**, 568.

——(1908). "Diseases of Occupation." London.

Purdy, J. S. (1903). *Brit. med. J.*, **1**, 1290.

Shufflebotham, F. (1914). *Brit. med. J.*, **1**, 588.

Smart, A. (1883). "Germs, Dust and Disease." Edinburgh.

——(1885). *Brit. med. J.*, **2**, 439.

Tatham, J. (1908). Supplement to 65th Annual Report of Reg.-Gen. for England and Wales, 1901. Part II. Cd. 2619. H.M.S.O. London.

Trotter, R. S. (1903). *Brit. med. J.*, **1**, 1197.

Brit. J. industr. Med., 1952, **9**, 99.

PNEUMOCONIOSIS OF COAL MINERS IN NORTH EAST ENGLAND WITH SPECIAL REFERENCE TO THE DURHAM COALFIELD

BY

R. I. McCALLUM

From the Nuffield Department of Industrial Health, University of Durham (King's College, Newcastle upon Tyne)

(RECEIVED FOR PUBLICATION FEBRUARY 2, 1952)

The true prevalence of coal miners' pneumoconiosis in north east England is unknown and this paper attempts to answer the following questions from existing evidence : (1) What is the past history of the disease in this area and how did it relate to mining practice? (2) What is the present prevalence and how does the type of disease vary with the age of the miner, and (3) is there a case for a pneumoconiosis survey in this coalfield?

From the earliest times coal has come from Newcastle (Nef, 1932). At the turn of the eighteenth century the introduction of the steam engine for pumping, winding, ventilation, and haulage led to the rapid expansion of the coal mining industry. The bord and pillar system, in which a series of narrow headings are driven into a seam parallel to one another and connected by cross headings, is more widely used in Durham than elsewhere, and mechanization has come more slowly here than in the rest of England (Smith, 1949).

John Evelyn, in his " Fumifugium " published in 1661, wrote that :

> " Newcastle Cole, as an expert Physician affirms, causeth consumptions, phthisicks and the indisposition of the lungs, not only by the suffocating aboundance of smoake, but also by its virulency, for all subterrany fuel hath a kind of virulent or arsenical vapour rising from it ; which, as it speedily destroys those who dig it in the mines, so does it, by little and little, those who use it here above them."

This suggests that at this time coal miners suffered from chest disease.

Gregory (1831) and Thomson (1837) first reported " black lungs " in Great Britain, but at this time only one proven case (Stratton, 1838) had been seen in Newcastle. In 1861 Sir John Simon mentioned that Northumberland and Durham miners were free from excess of pulmonary disease and suggested that this was due to good ventilation of their pits.

In 1866 and 1869 Greenhow demonstrated to the Pathological Society of London cases of colliers' black lung among miners from the Newcastle area. Oliver (1908) stated that cases of miners' phthisis were common in the Newcastle Infirmary towards the end of the nineteenth century, but he said that since the turn of the century very few cases had been seen. It appears then, that in the latter half of the nineteenth century what is now called the complicated form of miners' pneumoconiosis (Fletcher and Gough, 1950) was being recognized, but that at the beginning of the twentieth century it had almost disappeared. By the end of 1933 no miner from Durham had been certified as suffering from pneumoconiosis and only one from Northumberland, compared with 500 from Wales.

In 1943 the Workmens Compensation Act was extended to include earlier lung changes (dust reticulation). Table 1 (from McVittie, 1949) shows the number of men certified from 1939 to June, 1948, before the National Insurance (Industrial Injuries) Act took the place of the workmen's compensation acts, and demonstrates the great increase in certifications following the extended scope of this legislation.

The increase appears everywhere, but the total numbers in Northumberland, Durham, and Cumberland combined, are very small compared with Wales and the rest of the British Isles.

Figures published in 1948 by the National Union of Mineworkers (Durham Area), which, however, refer only to men whose application had been made through the Union, show (Fig. 1) an increase in the number of applications and successful claims between 1943 and 1948 in County Durham. Most of the men who made claims were face workers and half of them were successful.

TABLE 1

NEW CASES CERTIFIED BY THE SILICOSIS MEDICAL BOARD FROM 1939 TO JUNE 30, 1948*

Region	1939	1940	1941	1942	1943	1944	1945	1946	1947	Up to June 30, 1948
Scotland	7	2	3	6	10	109	148	166	322	175
Northumberland, Durham, and Cumberland	2	5	2	4	12	79	57	71	93	64
North Eastern (Yorkshire)	6	7	8	6	25	85	95	127	155	81
North Midland	—	1	—	1	4	10	13	25	17	10
North Western	10	10	8	7	27	53	81	97	136	62
Wales	418	448	499	763	1,155	1,608	5,224	3,804	2,867	1,069
Midland	12	11	13	24	48	95	132	99	110	64
Kent	10	—	2	10	25	30	71	51	79	59
Total	465	484	535	821	1,306	2,069	5,821	4,440	3,779	1,584

* From McVittie (1949).

The steady rise in the certification figures, notwithstanding their limitations, suggests a rise in prevalence or an increased local awareness of the disease, or a combination of both. During this period the greatest number of certified cases tended to be in the large modern pits near the coast, but collieries with a small number of certified cases were scattered widely over County Durham (Fig. 2). In Northumberland at the same time six men died of the disease and 41 were certified, most of them partially disabled.

The Recent and Present Position

Out-Patient Experience.—When the Nuffield Department of Industrial Health opened a clinic in Newcastle upon Tyne early in 1947 it was clear that a major local problem was the dyspnoeic coal miner. A study of the first 100 patients seen by Browne, Beck, and McCallum (1949) showed that 69 were coal miners, most of them with chest symptoms. Twenty-one men, all of them face workers, had pneumoconiosis, 16 with the simple and five with the complicated disease. The radiological changes were similar to those described in South Wales coal miners by Hart and Aslett (1942) and by Davies and Mann (1949).

Attention was further drawn to the problem when between March and July, 1948, 21 drillers from a Durham pit were examined at their own request because four of their workmates had recently been certified as suffering from pneumoconiosis. All of them had been drilling in both coal and stone for 13 to 32 years at the same pit. Fourteen men had abnormal chests. One man had pulmonary tuberculosis, probably active, and the remaining 13 pneumoconiosis, one of them the complicated type. Nine of these men were symptomless when first seen, and two of four men with shortness of breath and cough had had their symptoms over two years. Subsequently five of these drillers were granted compensation. Their ages ranged from 37 to 56 with an average of 49 years.

During the three years since the clinic was opened more than 300 coal miners with pneumoconiosis have been seen. Most of the men come from County Durham but there have been a few from Northumberland. A large number, many referred by their own doctors after mass miniature radiography, had no symptoms.

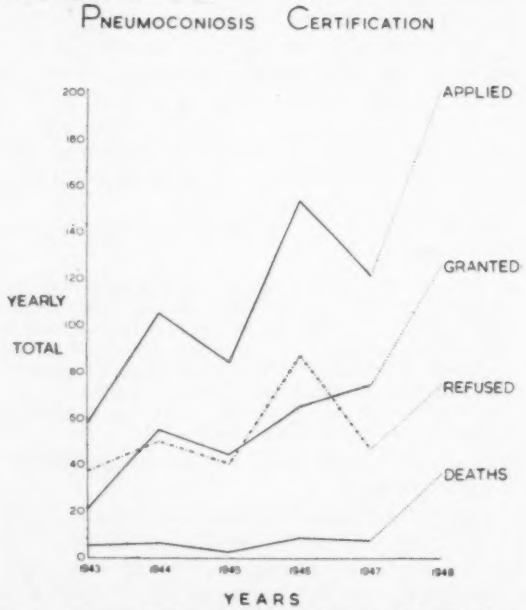


FIG. 1.—Pneumoconiosis certification in County Durham between 1943 and 1948 (July). As the figures for 1948 are available only up to July 5, the graph has been continued as if the same rate were maintained to the end of the year (dotted line).

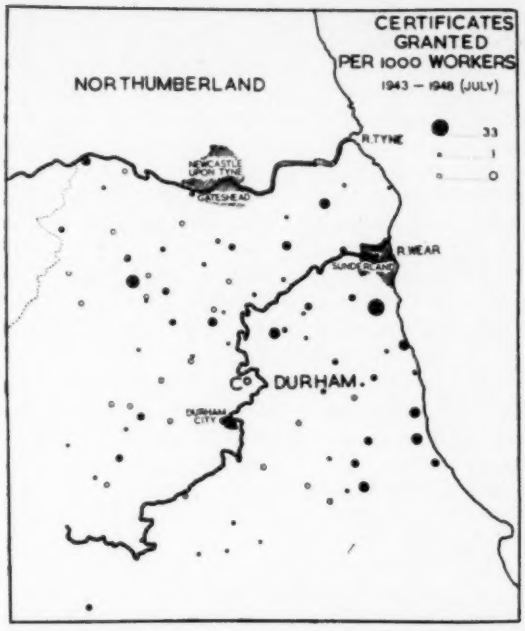


FIG. 2.—Distribution of certified cases of pneumoconiosis per 1,000 workers in the Durham coalfield in 1943-48.

The Limitations of Mass Miniature Radiography

Notwithstanding their limitations, the results of mass miniature radiography provide the only evidence of the prevalence of pneumoconiosis in this area, for the number of certified cases can be misleading in deducing the prevalence of pneumoconiosis in all its forms (Cochrane, 1951). These results should therefore be considered with reservations.

In August, 1948, a mass miniature radiography (M.M.R.) unit started work in the pits of County Durham. The examination was voluntary and primarily designed to detect cases of pulmonary tuberculosis. In the first year of its work eight pits were visited. It was soon obvious that pulmonary tuberculosis was not the main problem but that a number of miners had pneumoconiosis.

Response of Volunteers.—Forty-four to 61% of the men at the different pits visited volunteered for *x*-ray examination. It is difficult to say what determines who comes forward, because experience with out-patients shows that men with bronchitis and emphysema have similar symptoms to those of dust disease, while other men with simple or even complicated pneumoconiosis may have no symptoms at all. In some pits there was an atmosphere of suspicion lest the examination was designed to weed out men with "dust" with a view to discharging them, despite the repeated assurance that the examination was confidential. Thus some men who thought they had pneumoconiosis may have been discouraged from attending and others who thought they were in good health may have considered that it was unnecessary for them to be examined.

The ages of all men attending the M.M.R. unit for a miniature film at three collieries between March and August, 1949, are known, and the distribution of age was compared with that of all the men in each of the three collieries, using figures obtained from the National Coal Board of a manpower survey made in December, 1948. It was assumed that the age pattern would not have altered materially over a few months (Table 2). In this table there is no systematic change in the percentage of volunteers with age; but as age increases there is an increase in dyspnoea from all causes and a presumed increase in pneumoconiosis. It is unlikely, therefore, that the presence of dyspnoea

TABLE 2

NUMBER OF MEN IN EACH OF THREE COLLIERIES ATTENDING FOR MINIATURE FILMS COMPARED BY AGE TO TOTAL NUMBER OF MEN ON COLLIERY BOOKS

Age	Colliery E			Colliery F			Colliery H		
	No. of Men	Miniature Films		No. of Men	Miniature Films		No. of Men	Miniature Films	
		No.	%		No.	%		No.	%
19 and under	147	47	32	163	62	38	156	62	40
20–29	347	210	61	429	220	51	338	164	49
30–39	289	185	64	440	246	56	445	199	45
40–49	277	196	71	460	292	63	430	260	60
50–54	107	70	65	170	95	56	138	106	77
55–59	88	55	63	171	76	44	104	57	55
60 and over	80	48	60	141	72	51	125	42	34
Total	1,335	811	61	1,974	1,063	54	1,736	890	51

or pneumoconiosis has influenced volunteering to any great extent.

Diagnosis from Miniature Films.—Another problem was the unknown number of men with early degrees of pneumoconiosis who might be overlooked in viewing the miniature films. At each pit the men were first examined by miniature films which were viewed by the director of the M.M.R. unit, who had no special experience of pneumoconiosis. Those thought to be abnormal in any way were recalled for a large film. It was unlikely that he was able to detect Category 1 changes from a miniature film. Category 2 changes might or might not be detectable on certain miniature films ; and only for Category 3* changes could a definite opinion be given that pneumoconiosis was present. Many lungs which appear abnormal on miniature films because of prominent vascular shadows are found to show no pneumoconiosis when a full size film is taken, and others might show early opacities due to dust. The difficulties of classifying the large films are sufficiently great to make classification of small films a waste of time.

A test was carried out with 102 consecutive miniature films of miners at the same pit and large films of the same men. The miniatures were classified by one observer and the results compared with a classification of the large films by the same observer reading with another observer using a standard set of categorized films. It will be seen from Table 3, assuming that the reading of the large films was correct, that 35 miniatures were placed in too high a category, 27 in too low a category, and in only 40 was there agreement. Also, of the 48 cases with normal large films (Category O), 19 were placed more than one category higher in the corresponding miniature films. Thus the tendency was to read higher on the miniature films, and it appears that miniature films are likely to be misleading in a survey to detect simple pneumoconiosis in all its stages.

* These categories are fully described by Davies and Mann (1949).

Experience in South Africa, where mass miniature radiography has been used extensively for native labourers, has been that miniature films tend to exaggerate abnormal shadows such as tubercular lesions but that there is difficulty in reading the miniatures of early silicosis (Retief, 1946). It appears that only full size films are likely to be of use in detecting pneumoconiosis in coal miners, where the choice lies between them and the miniature (35 mm.) film. We have, therefore, not used the miniature films for categorization. Only the large films have been considered. These were only available in a small proportion of the cases—about 10% of the total number of men in the pits.

Viewing.—Two observers examined the large films on a standard viewing box. Sessions were kept short to avoid fatigue and lasted from three-quarters to one hour, and the final category had to be agreed by both observers. Where there was doubt, a number of reference films which had been classified by members of the Pneumoconiosis Research Unit in Cardiff, using their system (Fletcher, Mann, Davies, Cochrane, Gilson, and Hugh-Jones, 1949), were used as a standard (Fletcher and Oldham, 1949, 1951). These reference films had been taken at a Durham colliery with the same machine and technique as that used for the films being classified. A copy of Davies and Mann's (1949) paper, which describes each category, was also available for reference. One of the two observers had visited the Cardiff unit and had seen the method in use.

Each film was viewed without knowing the man's age or occupational history, and the category was entered on to his history sheet by a clerk to eliminate bias from a knowledge of the working history. With good quality films there was usually no difficulty in classifying from Category 3 upwards, but the variation within Categories 1 and 2 appears to be rather greater than in the higher categories and small differences in film quality made classification more difficult and less consistent. This was

TABLE 3

COMPARISONS OF READINGS OF LARGE AND MINIATURE FILMS OF THE SAME SUBJECTS AT A DURHAM PIT

	Category	Miniature Films : Observer B Category					
		0	1	2	3	4	Total
Large Films : Observers A and B	0	25	4	18	1	—	48
	1	10	1	6	3	—	20
	2	2	—	7	3	—	12
	3	1	—	12	7	—	20
	4	—	—	—	2	—	2
	Total	38	5	43	16	—	102

intensified by the difficulty encountered with fluctuations in current supplies at some collieries, resulting in underpenetrated films.

Amongst the most difficult tasks in using the Davies and Mann classification was the differentiation between a normal film and the earliest detectable changes due to dust, Category 1. This was particularly hard in the somewhat soft films used in these surveys as the normal vascular shadows tended to obscure the smaller details of the lung fields. For this reason, and as a check on the general suitability of the classification for use in coalfields other than South Wales and by different observers, a comparison was made between two readings by Newcastle observers and two readings by members of the Pneumoconiosis Research Unit in Cardiff of the same set of 64 large films taken by the Durham M.M.R. unit and selected from Categories 0 to 2. Two of these films were rejected as unreadable by the Cardiff observers and have not been included in the results in Table 4. These readings showed that the Newcastle observers tended to place the films in somewhat higher categories and were less consistent than the Cardiff observers, but the difference was not gross.

TABLE 4

COMPARISON OF READINGS* OF 64 LARGE FILMS BY CARDIFF (P.R.U.) AND NEWCASTLE OBSERVERS

Category	Newcastle Observers		Cardiff (P.R.U.) Observers	
	A & B	A & C	D	E
0	31	27	38	40
1	31	27	23	20
2	0	8	1	2
Mean category	0·50	0·69	0·40	0·39

* Films were read twice in each centre. Cardiff observers read the films on separate occasions, Newcastle observers in pairs, one observer reading on both occasions.

Evidence from Mass Miniature Radiography.—One thousand four hundred and sixty-eight large films from eight Durham pits were viewed and classified (Table 6). Six of the pits were in the eastern part of the coalfield and the others in the west and south. Two of the six are coastal and work undersea coal. They were not specially selected but were taken in order, as each request for a visit came from the men. The large films showing pneumoconiosis have been divided into two groups, those showing Category 1 changes only and those showing more advanced changes. This has been done because of the greater disagreement we have found among our observers in placing a film in Category 0 or 1. The nature of the sample of miniature film is not accurately known and the errors in selecting men for large films have already been referred to, so that the number of men with pneumoconiosis has been expressed not only as a

TABLE 5

READINGS OF MIXED SERIES OF FILMS BY CARDIFF (P.R.U.) AND NEWCASTLE OBSERVERS

Simple Categories

Cardiff \ Newcastle	0	1	2	3	4	Total
0	5	2	—	—	—	7
1	1	2	—	—	—	3
2	—	—	2	4	—	6
3	—	—	—	14	2	16
4	—	—	—	—	2	2
Total	6	4	2	18	4	34

Newcastle high by one category	8
Newcastle agree	25
Cardiff high by one category	1
	34

*Complicated Categories**

Cardiff \ Newcastle	Nil	A	B	C	D	Total
Nil	—	2	—	—	—	2
A	—	3	—	—	—	3
B	—	1	1	—	—	2
C	—	—	—	1	—	1
D	—	—	—	—	—	—
Total	—	6	1	1	—	8

Newcastle high by one category	2
Newcastle agree	5
Cardiff high by one category	1
	8

*Each complicated category is also classified 1, 2, 3 or 4. Progressive massive fibrosis (A to D) has been considered separately.

A further comparison was made between readings of a mixed series of films covering all the simple categories and complicated Categories A to C. There were 34 films and each was classified once in Cardiff and once in Newcastle. Again the Newcastle readings were somewhat higher but there was better agreement in the small number of complicated categories (see Table 5).

TABLE 6

NUMBER AND PROPORTION OF MEN WITH PNEUMOCONIOSIS IN EIGHT PITS IN COUNTY DURHAM

Pit	Estimated No. of Men Above and Below Ground Dec., 1948	No. of Miniature Films Taken (Sample)	Large Films			Percentage Above and Below Ground with Pneumoconiosis			
			Total Viewed	Pneumoconiosis		Cat. 1 Only		Cat. 2 and Over	
				Cat. 1 Only	Cat. 2 and Over	In Sample	Total Men in Pit	In Sample	Total Men in Pit
A	1,775	919	159	26	68	2·8	1·5	7·4	3·8
B	2,659	1,346	301	75	96	5·6	2·8	7·1	3·6
C	1,858	1,057	98	14	35	1·3	0·75	3·3	1·9
D	2,566	1,137	273	80	61	7·0	3·1	5·4	2·4
E	1,335	811	170	39	41	4·8	2·9	5·1	3·1
F	1,974	1,059	165	32	35	3·0	1·6	3·3	1·8
G	1,460	715	147	27	61	3·8	1·8	8·5	4·2
H	1,736	893	155	25	55	2·8	1·4	6·2	3·2
Total	15,363	7,937	1,468	318	452	4·0	2·1	5·7	2·9

percentage of the sample but also as a percentage of the total number of men at the colliery. The latter figure represents, therefore, a minimum one.

Assuming that all the films read as Category 1 were in fact normal, at least 1·9% to 4·2% of all the men at these eight pits have pneumoconiosis (Category 2 and over), or between 3·3% and 8·5% of the sample attending for a miniature film. These are the men with really significant changes. The most conservative estimate of pneumoconiosis, all categories, at these pits would then be between 3% and 6% of the total working population. It is impossible to estimate the disability associated with the disease in these men as only a small proportion were examined clinically. However, most of those who were radiographed were actively at work at the time, and our out-patient experience shows that there is little relation between disability and the *x*-ray appearance.

Radiological Type of Disease.—Most of the pneumoconiosis seen in these films was of the simple variety, mainly in the first three categories. Category 4 was not found at all frequently. The number of films placed in each category, and the number considered normal (Category 0), is shown for each pit in Table 7 which shows that the complicated variety is much rarer and is represented in these films mainly in Category A with a few in Categories B and C. There are no films in Category D, but we have seen men in this category in our out-patient work, though infrequently. In some cases it has been possible to pick out films which in appearance suggested classical silicosis (second stage) rather than coal workers' pneumoconiosis, and usually on reference to the occupational history a long period of stone drilling has been noted. These films have not been included in the present series.

TABLE 7

LARGE FILMS TAKEN AT EIGHT DURHAM PITS CLASSIFIED BY PIT AND CATEGORY

Category		Pit																Total	
		A		B		C		D		E		F		G		H			
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Simple	0	65	41	130	43	49	50	132	48	90	53	98	59	59	40	75	48	698	48
	1	26	16	75	25	14	14	80	29	39	23	32	19	27	18	25	16	318	22
	2	26	16	53	18	7	7	43	16	26	15	27	16	31	21	29	19	242	16
	3	21	13	32	11	13	13	14	5	8	5	6	4	20	14	18	12	132	9
	4	5	3	2	1	4	4	1	—	4	2	1	1	0	—	0	—	17	1
Complicated	A	10	10	8	3	8	11	2	1	3	2	1	1	3	7	7	5	42	4
	B	5		0		3		1		0		0		6		1		16	
	C	1		1		0		0		0		0		1		0		3	
	D	0		0		0		0		0		0		0		0		0	
Total		159	99	301	101	98	99	273	99	170	100	165	100	147	100	155	100	1,468	100

TABLE 8

NUMBER AND PERCENTAGE OF MEN IN EACH RADIOLOGICAL CATEGORY BY AGE

Age in Years	Category					Total	Percentage in Cat. 1 and Over	Percentage in Cat. 2 and Over
	0	1	2	3	4			
14–18	22	—	—	—	—	22	0	0
19–23	45	2	—	—	—	47	4	0
24–28	76	16	4	1	—	97	22	5
29–33	68	19	7	5	—	99	31	12
34–38	120	40	36	22	3	221	46	28
39–43	99	58	56	31	5	249	60	37
44–48	79	74	38	28	6	225	65	32
49–53	76	50	50	37	4	217	65	42
54–58	67	25	29	20	4	145	54	37
59–63	30	24	25	18	1	98	69	45
64–68	8	5	1	6	1	21	62	38
Total	690	313	246	168	24	1,441	52	30
Average age	41	46	48	49	48	45	—	—

Age and Category

When the men with pneumoconiosis are considered by age and category (Table 8) it is seen that, excluding two men in Category 1, all categories occur in men over 24 years of age. Nearly all the men with pneumoconiosis will have done at least six to eight years' underground work which would include four to five years' pneumatic hewing. Below 30 years of age there are few men in Category 2 or 3. Dust changes are more common after the age of 34 years. After 39 to 44 years there is no marked change in the percentage of men with abnormal films in each five-year age group. This may be because men with more advanced pneumoconiosis tend to leave the pit after this age, or because the older men are changing to less dusty work away from the face, for the average age in each category does not vary very much. It is noteworthy that after the age of 54 years about a third of the men show no recognizable dust changes on radiography. The occupational histories of these men did not differ from those who had pneumoconiosis, a fact which suggests a difference in susceptibility.

Discussion

It is clear from the evidence presented here that there is a fairly widespread prevalence of lung changes due to dust, particularly in the Durham coalfield and probably to a lesser extent in Northumberland. It must be emphasized, however, that so far it appears that the north east reflects only faintly conditions in South Wales, and not only are the numbers of miners affected far smaller than in Wales but also the disease is probably less severe, with perhaps less risk of superimposed tuberculous infection. Nevertheless the pathology of pneumoconiosis in the north east is similar to that in the soft coal areas of South Wales (Duguid, personal communication). In the 40 years since Oliver's reference to the disease there has been no further mention of it in the north east other than a suggestion that there may have been a true increase in incidence of the disease in other coalfields than the South Wales one, though not such a serious increase (Fletcher, 1948).

There may well have been a small incidence of pneumoconiosis in this area after the end of the nineteenth century, but a black lung or "anthracosis" in a coal miner is often accepted as a normal finding, and the complicated form may have been regarded as purely tuberculous. From the clinical standpoint many miners who were, in fact, suffering from the effects of dust inhalation have in the past undoubtedly been considered to have bronchitis, or bronchitis and emphysema. The limitations of ordinary chest radiology have not been sufficiently appreciated, because it has only recently been shown how much individual opinions may differ even where the radiography is of the highest quality (Fletcher and Oldham, 1949). But it is unlikely that a high prevalence of gross pathological change in the lungs of miners in the north east would have passed unnoticed, particularly in institutions such as the Royal Victoria Infirmary in Newcastle upon Tyne where a fifth of adult male admissions are coal miners.

Greenhow's (1866, 1869) and Oliver's (1908) description of black lungs in the north east between 1860 and 1880 were after the modern development of the Durham coalfield and its greatest expansion.

But at the same time improvements in ventilation were being introduced, largely because of mine gases and the risk of explosion. Because of the long natural history of pneumoconiosis it seems reasonable to link these facts and to correlate the early expansion of the coalfield with the evidence of gross pneumoconiosis 20 or 30 years later. This was before mechanization, and only the hand pick was used in the coalfield, so that ventilation was probably the main factor involved. The improvements in ventilation which followed closely deeper and more extensive mines probably sufficed to reduce dust concentration to a level low enough to make pneumoconiosis a rarity for the time being. It seems likely that increased dustiness from mechanization has reversed this trend over the last 30 years, and that ventilation has not always kept pace with the increase in the depth and extent of the pits.

Although relatively a small problem compared with that in some of the other coalfields in Britain, pneumoconiosis in Durham is now of considerable local importance and its full implications must still be assessed. The present data, derived from the work of a mass radiography unit in certain pits in Durham, despite their limitations, give the most complete information which is at present available.

Conclusion

There seems to be no reason to fear the results of pneumoconiosis surveys, because the miners themselves know that the disease occurs, and on the whole welcome an attempt to investigate it. Mass radiography in a mining area must be prepared in advance to deal with pneumoconiosis. In Durham it was arranged that men could be referred to the Nuffield Department of Industrial Health through their own doctors, if further advice was required. In advising men with pneumoconiosis, an attempt is made to counteract the psychological ill-effects of the diagnosis by explanation and reassurance.

As these results suggest that the Newcastle observers have tended to read too high, though not consistently, the minimum figures for the total of all categories at each pit may be considered to be too high and misleading. Films placed in Category 1 and also some in Category 2 would probably be found to include some normal films if they were re-read, or read by different observers. The present definition of the lower limit of simple pneumoconiosis may need to be revised, as even skilled observers occasionally diagnose early Category 1 changes in men without dust exposure (Fletcher and others, 1949). A more accurate assessment might be made by increasing the number of observers or observations, that is by re-reading the films until a consistent result was obtained. The latter requires great time expenditure and the former a considerable number of experienced staff. In both cases films of first class quality are desirable.

It is clear that complete surveys of several representative pits throughout the coalfield, in which all the working population or at least all the underground population are examined with large films, are required to amplify these figures, and one such survey has by now been carried out. Preliminary results suggest that the figures in this paper underestimate the prevalence of pneumoconiosis. Miniature mass radiography is of doubtful value as far as pneumoconiosis is concerned, as it will only detect the grosser forms with certainty and gives little idea of the prevalence of simple dust retention in the men's lungs. From the preventive aspect it is important to gain a complete picture of the prevalence at a particular pit and at present it appears that this can only be done by using full size *x*-ray films. On the other hand, when a visit from the M.M.R. unit has been requested by men at a colliery this should be made as for other industrial groups, and every effort made to detect cases of open tuberculosis in view of the relation between this disease and pneumoconiosis (Mann, 1951).

The evidence so far suggests that the larger and deeper coastal pits have been very dusty during the past two or three decades and any further mechanization or any falling off in the efficiency of their ventilation is likely to make them dustier still. A higher incidence of pneumoconiosis will be the inevitable result unless dust suppression (already in use in some Durham pits) is further extended, and it will be required in all pits which are actively developing and ultimately in all faces where any form of mechanized coal-getting is used.

Summary

Pneumoconiosis of coal miners has, in the past, occurred in a gross form in north east England following a period of expansion in the coal industry. Improvement in ventilation probably reduced the clinically recognizable disease to small proportions towards the end of the nineteenth century and beginning of the twentieth.

Mechanized coal-getting has probably increased the true prevalence of pneumoconiosis, but diagnosis has also improved. The changes in the lungs of Durham miners appear to be similar to those found in the soft coal area of South Wales except that they are less severe.

Evidence from routine mass miniature radiography at eight Durham pits suggests that a minimum of 3% to 6% of men in these pits show well marked pneumoconiosis.

Future developments in the coalfield will tend to

intensify dust production and the risks of pneumoconiosis unless dust suppression methods are considerably extended.

Acknowledgments are due to Professor R. C. Browne for help with this paper ; to Dr. D. M. Calvert, Medical Director, Mass Radiography Unit, Durham, and the Regional Hospital Board, No. 1 Region, for their cooperation in obtaining and viewing films, and to the staff of the unit for their help in obtaining occupational histories ; also to the National Union of Mineworkers, Durham Area, for their assistance, and to Mr. H. Campbell for statistical help.

REFERENCES

Browne, R. C., Beck, I. F., and McCallum, R. I. (1949). *Brit. J. soc. Med.*, **3**, 77.
Cochrane, A. L. (1951). (Proceedings of Conference) " The Application of Scientific Methods to Industrial and Service Medicine," p. 97. Med. Res. Coun. H.M.S.O. London, March 1950.
Davies, I., and Mann, K. J. (1949). *Proc. 9th Int. Congr. Industr. Med.* London. 1948, p. 768. Bristol.
Duguid, J. B. Personal communication.
Evelyn, J. (1661). " Fumifugium." Colburn, London, 1825.
Fletcher, C. M. (1948). *Brit. med. J.*, **1**, 1015, 1065.
——, and Gough, J. (1950). *Brit. med. Bull.*, **7**, 42.
——, and Mann, K. J., Davies, I., Cochrane, A. L., Gilson, J. C., and Hugh-Jones, P. (1949). *J. Fac. Radiol., Lond.*, **1**, 40.
——, and Oldham, P. D. (1949). *British Journal of Industrial Medicine*, **6**, 168.
——, and Oldham, P. D. (1951). *Ibid.*, **8**, 138.
Greenhow, E. H. (1866). *Trans. path. Soc. Lond.*, **17**, 36.
——(1869). *Ibid.*, **20**, 41.
Gregory, J. C. (1831). *Edinb. med. surg. J.*, **36**, 389.
Hart, P., D'Arcy, and Aslett, E. A. (1942). *Spec. Rep. Ser. med. Res. Coun. Lond.*, No. 243.
Mann, K. J. (1951). *Thorax*, **6**, 43.
McVittie, J. C. (1949). *Postgrad. med. J.*, **25**, 618.
National Union of Mineworkers (1948). General Secretary's Annual Report. (Durham Area.)
Nef J. U. (1932). " The Rise of the British Coal Industry." London.
Oliver, T. (1908). " Diseases of Occupation." (3rd Ed. revised 1916). London.
Retief, F. (1946). *Proc. Transv. Mine med. Offrs' Ass.*, **26**, 39
Simon, J. (1862). *4th Rep. med. Offr. Privy Coun., Lond.*, 1861. P. 21.
Smith, W. (1949). " An Economic Geography of Great Britain." London.
Stratton, T. (1838). *Edinb. med. surg. J.*, **49**, 490.
Thomson, W. (1837). *Med-chir. Trans.*, **20**, 230. (Abstract in *Edinb. med. surg. J.*, 1838, **49**, 260.)

Brit. J. industr. Med., 1952, **9**, 108.

THE MECHANISM OF DISPERSION OF COAL PARTICLES IN THE LUNGS OF MINERS

BY

A. POLICARD

From the Centre d'Études et de Recherches des Charbonnages de France, Paris

(RECEIVED FOR PUBLICATION JANUARY 15, 1952)

Coal particles inhaled by miners in the course of their work, especially those of 2 microns in diameter and less, are carried as far as the alveoli. Particles which have reached the alveoli and become attached to their surface are phagocytosed by the alveolar cells, and in this way dust cells are formed. This is the first stage in the migration of coal-dust, of which the second is its deposition in the peri-arterial and peribronchial connective tissue sheaths, as well as in certain other fibrous tissues such as the pleura. Some particles can also travel even further, into the tracheo-bronchial glands and beyond, but these will not be considered here.

This paper is concerned with the histological and physiological mechanisms of dust transportation and the pathways along which dust cells travel. It is a particularly complicated problem, because animal experiments cannot give us reliable information as the behaviour of the lungs is different in each species. The lungs of a rabbit do not behave in exactly the same way as those of a rat, still less as those of a man. Extrapolation from animal species to man is therefore not valid, as I have emphasized elsewhere (Policard, 1950). I need not go further into the question here. The only acceptable observations, therefore, are those made on man himself but there other difficulties arise, of which the most serious is that when the thorax is opened for removal of the lungs, they collapse. This collapse of the lung, which is accentuated by the action of fixatives, upsets normal relationships. The contraction does not produce lesions but changes the topographical relationship of the structures. It would be best to fix the lungs in position without opening the thorax, but although this is theoretically possible, it would not be practicable in the case of man. It is possible, however, to inflate the lungs before fixing them, although it is difficult under the conditions of ordinary hospital necropsies. Moreover, this procedure does not usually give good results because of the post-mortem fragility of the alveolar walls, which rupture on inflation. Under normal post-mortem conditions, one can only obtain a lung which is not collapsed when the alveoli of part of the lung are filled with a transudate, as in a case of acute oedema of the lung, or blood, as in the case of pulmonary infarct; for then, this part of the lung does not collapse when the thorax is opened and the alveoli are still in their normal position of distention even after fixation. It is a case of this kind which constitutes the basis of this paper.

Case Report

A miner aged 40, who had worked in coal for 20 years and was in excellent health, was caught while at work in coal dust by an escape of fire-damp (methane) and collapsed with asphyxia. (Methane has no toxic effect on pulmonary tissue but kills by simple asphyxia.) The miner was removed unconscious after some minutes and taken to the surface. Shortly afterwards he showed signs of acute pulmonary oedema (anoxic oedema) and died two hours after the accident. For medico-legal reasons the necropsy was carried out five hours after death, that is seven hours after the beginning of asphyxia. The lung showed the usual appearance of acute oedema and in particular it showed no collapse.

The interest of such a case, as far as it concerns our problem, is (1) that the man was struck down suddenly while at work and while actually breathing coal dust. (2) The additional process of passive oedema prevented the collapse of the lung that usually occurs after death and so avoided displacement of pulmonary structures. (3) The tissues were fixed a relatively short time after death and so allowed histological examination to be carried out under good conditions.

A histological study of this case has shown the usual appearances seen in coalworkers' lungs such as have been described by Gough (1947) and by Heppleston (1951): the presence of an accumulation

of dust cells in the peri-arterial and peribronchial tissues, which are hypertrophied and fibrosed, though not to a very great extent (these accumulations constitute the macules of the Welsh workers), and clear evidence of a certain amount of focal emphysema of the type described by Gough (1940, 1947). In this lung the following histological observations could be made.

Dust in the Alveoli Phagocytosed by Alveolar Cells

In the alveolar cavities, well dilated by the presence of the oedematous exudate, there were practically no free particles but, on the other hand, numerous alveolar cells stuffed with coal particles, the cells being free in the transudate or attached to the alveolar wall. The rarity of free particles can be easily understood. The transudation of blood plasma through the capillaries, resulting from the development of oedema, has probably carried away the particles which were on the alveolar walls. These particles could also have been phagocytosed during the period of formation of oedema, the exudate constituting a kind of culture medium in which the phagocytes could operate. However it may be, these observations do not allow us to draw any conclusion about the presence of free particles in the alveoli ; the data are only applicable to dust cells.

The dust cells found in the alveoli were quite typical. They were rounded or ovoid structures, 18–20 μ in diameter, with clearly defined borders but no sign of a membrane. They contained an ovoid or kidney-shaped nucleus, 6–8 μ in diameter, with a fine chromatin network. This arrangement is well known.

The arrangement of the coal particles was variable and two chief types of cells could be distinguished. In some the particles, which were few, were well spaced ; sometimes there were two or three to the cell, sometimes 10 to 20. They were situated in the cytoplasm, generally close up against the nucleus. The latter was not modified by their presence. No cells were to be seen in the periphery of the cytoplasm. In the other type of cell the particles were closely packed and were situated in the centre of the cell in contact with the nucleus.

Observations on man differ somewhat from those made in animal experiments. In the latter the cells always enclose a very large number of particles, never two or three. This suggests that isolated particles are not phagocytosed by the cell, which only picks up aggregates of particles formed outside it and ingests them *en bloc*. This process, often clear in the animal, does not seem to be the only one, phagocytosis of single particles being also possible. This is a question which requires further study.

The particles seen were entirely carbonaceous. In polarized light practically no birefringent particles were seen. They were all black or brown and composed of coal. Most of them were between 0·4 and 0·8 μ in diameter. Particles from 1 to 2 μ were much less frequent and only occasionally particles from 3 to 8 μ were seen.

The dust cells were usually free in the liquid which filled the alveolar cavities. A few, however, were still attached to the alveolar walls. Besides these dust cells one occasionally found alveolar cells of the same type but containing no dust. No dust cells were seen showing signs of degeneration and cytolysis, that is to say distended and more or less disrupted.

Dust cells so formed in the alveoli pass into the interstitial tissue and then take several courses. Two points must be considered here ; the route by which they are removed, and the mechanism by which it is carried out, whether passive translation by the current of lymph or active amoeboid movement. It is obvious that as no direct demonstration of these movements was possible, any answer to these questions can only be indirect deductions from morphological observations, and they are correspondingly uncertain.

Apart from those dust cells which reach the bronchioles, and hence the bronchi and trachea, one must consider two chief routes of penetration into the interstitial tissue, and especially into the peri-arterial and peribronchial sheaths ; the route through the terminal bronchioles and the route through the periphery of the sheaths.

Penetration of Dust Cells at the Level of the Terminal Bronchioles

At the end of the terminal bronchioles there is a point of transition to the alveolar duct, a position of histological and physiological importance (Policard and Collet, 1951). At this level the bronchial epithelium ceases and the connective tissue of the bronchial sheath is in contact with the alveolar air and is not covered with epithelium. Dust cells coming from the alveoli are carried towards the bronchi, in the thin layer of liquid which covers the surface of this region. At the point where the bronchial epithelium begins, the cells can take one of two routes. They can pass over the epithelium and so reach the bronchi and be removed by expectoration, or they can pass under the epithelium and embed themselves in the connective tissue between its collagen fibres. In this way these cells, whether of their own accord or translated passively by the current of lymph, are carried to the peribronchial interstitial tissue. The dust cells can also attain the adjacent peri-arterial connective tissue,

with which the latter tissue is always continuous. By their accumulation at certain points, the dust cells form the peribronchial and peri-arterial nodules which can be seen in lung sections in coal-workers' pneumoconiosis.

The mechanism by which particles penetrate to the end of the bronchioles is only a particular case of a general process. It also occurs with micro-organisms, as has been known for a long time.

The same movement of dust cells has often been observed in animal experiments. On the other hand, it is very difficult to observe it in man because of the disturbance of the lung produced by post-mortem collapse. In the case we have studied it was easily observed.

Penetration of Dust Cells into Perivascular and Peribronchial Tissue by the Peripheral Route

Dust cells in the alveoli can also get into the peri-arterial and peribronchial sheaths from their periphery. The mechanism seems to be as follows. The peri-arterial and peribronchial sheaths are surrounded by alveoli. At this level, as in all alveoli, this sheath is in contact with the alveolar surface. There is no continuous epithelium at this level but only a very incomplete layer of alveolar cells capable of phagocytosis. By their own movement, and also because of the flow of lymph from their surface towards the bronchi, dust cells can penetrate into the spaces between the fibres of the perivascular and peribronchial sheaths.

The following points should be noted. Dust cells can penetrate into the sheath from the periphery at all points but not always with equal ease. There are points where penetration is quite easy and there one finds a large number of cells. This probably explains the irregular form of the anthracotic nodule in the lungs of coal miners. The reason for this difference is still unknown but it seems possible that it depends on whether the fibrous tissue is closely or loosely packed.

Many anthracotic nodules, in fact most, are surrounded by emphysematous alveoli (focal emphysema of Gough). The presence of this emphysema does not seem greatly to affect the penetration of dust cells at these points. Often around certain partially developed anthracotic nodules, alveoli can be seen in a state of collapse. This condition seems to favour peripheral growth of the nodule. The alveolar walls collapse against the sheaths, increasing their diameter. In becoming attached and more or less fused to them, the alveolar walls enclose the dust cells which lie inside the alveolar cavities. This method of growth of anthracotic nodules seems to be common and important and is one which has been generally neglected. It is not seen in animal experiments but is very clearly seen in man under favourable conditions.

Often an accumulation of dust cells is seen in the lymph channels as numerous as in the connective tissue and, as a result of this accumulation, there is lymphatic obstruction. This suggestion, although fairly well founded, calls for certain reservations. In many places dust cells are found outside but not within the connective tissue lymphatics. This is particularly well seen in an undeveloped anthracotic nodule. The central artery shows a loose layer of connective tissue which isolates it from the pulmonary tissue (mobile layer of Von Hayek) and which contains the lymphatics. In the large nodules, on the other hand, the layer of dust cells extends up to the media of the artery. In certain cases also, in the most advanced stages of pneumoconiosis, dust cells may be seen in the media itself, which in this event is always abnormal. This suggests that the dust cells first arrive at the periphery of the sheaths. Particles which come via the lymphatics are carried by the lymph stream beyond the lung into the tracheo-bronchial glands. Interference with lymphatic function is a late phenomenon. Only then do the dust cells begin to accumulate in the loose inner layer of the connective tissue sheath.

These observations are of interest in so far as they concern the origin of vascular disturbances which develop in advanced coalworkers' pneumoconiosis.

Characteristics of Coal-dust Cells in Anthracotic Nodules

When they are free in the alveoli, coal-dust cells are regular spherical or ovoid structures but when they are in the fibrous tissue sheaths they have a different appearance. They are very irregular and are distorted by the surrounding connective tissue fibres. They behave like all tissue cells, whether fibroblasts or histiocytes, their shape depending on the more resistant neighbouring structures. In accordance with the arrangement of these surrounding structures, the dust cells are elongated or flattened but relatively much less than normal connective tissue cells which do not contain particles. The presence of hard particles prevents the cells from being deformed beyond a certain limit. Moreover, in cross section, the sheaths themselves are star-shaped.

In anthracotic nodules the cells are cytologically much altered. They have degenerated and their nucleus has usually disappeared. Their cytoplasm is hardened into a sort of cement (Policard and Collet, 1951) which encloses the particles and keeps them aggregated. This degeneration occurs by a fairly characteristic kind of dessication. One never

sees coal-dust cells distended or disrupted by proteolysis. This suggests that, by a mechanism as yet unknown, the coal particles inhibit the proteolytic enzymes normally present in the cells.

Fate of Collapsed Alveoli at the Periphery of Anthracotic Nodules

The walls of the collapsed alveoli surrounding the sheaths and the anthracotic nodules are joined together and seem to fuse to one another to form a fibrous mass. The reality of this fusion appears to be undoubted but its mechanism remains obscure. The following hypothesis is possible. The collapse of the alveoli leads to circulatory disturbances with the formation of an exudate into what remains of the alveolar cavities, thus distending the alveolar walls. This produces some repair of the connective tissue of the walls with a formation of new collagen ; initially, as usual, by precollagenous or reticulin fibres. Little by little the reticulin fibres are converted, as is customary, into true collagen fibres.

The part of alveolar collapse in the fibrous thickening of the sheaths from the outside involves the role (often discussed in pulmonary pathology) of atelectasis in the genesis of certain pulmonary fibroses.

The Passage of Single Particles in the Alveoli into the Peri-arterial Sheaths

It may be asked whether coal particles can be carried singly by the lymph and so reach the sheaths without being carried by cells. Once in the sheaths they could be phagocytosed by histiocytes present at that point.

In the course of the present investigation nothing has been seen which leads to a belief in the existence of such a process. In the peri-arterial sheaths particles are always collected in cells in a mass of the shape of the dust cell. It is true that it is important not to consider as particles of independent origin those which have clearly been displaced by the action of the microtome knife. This cause of error is well known but it is not always easy to avoid. On the other hand, in the sheaths in their normal state sufficient histiocytes are not found to explain the presence in this region of the large number of dust cells which are found there. Without actually denying the possibility of the transport of free particles from the alveoli to the sheaths, it seems it must be admitted that if this process exists it is most exceptional.

Conclusions

With the reservations which are necessary when making physiological deductions from histological observations, it seems that it may be suggested that the anthracotic nodule so characteristic of the lung of coal miners could be produced in the following way.

Two processes are involved : a proportion of the dust cells formed in the alveoli reach the peri-arterial and peribronchial connective tissue by two different routes. Some dust cells enter the pulmonary connective tissue at the level of the terminal bronchioles and reach the lymphatic channels normally present in the sheaths. So long as the sheaths are not abnormal, the dust cells are carried by the lymph and pass to the tracheo-bronchial glands. Other dust cells formed in the alveoli adjacent to the sheaths penetrate them from outside. They are found in the periphery of the sheaths. It seems that the dust cells remain stationary and are not carried to the lymph nodes.

The anthracotic nodule appears to result from the growth of sheaths thickened in the following way. At the periphery of the sheaths the alveoli collapse. Their walls are applied to the external surface of the sheaths which is thickened by them. The dust cells which happen to be in the alveolar cavities are thus included in the thickened wall of the sheaths, that is to say, in the outer part of the anthracotic nodule. The dust cells thus included in the nodule seem destined to remain permanently fixed there.

In the sheaths which have been progressively thickened and changed into anthracotic nodules some changes in the connective tissue occur, a process which is being actively studied at present.

References

Gough, J. (1940). *J. Path. Bact.*, **51**, 277.
——(1947). *Occup. Med.*, **4**, 86.
Heppleston, A. G. (1951). *Arch. industr. Hyg.*, **4**, 270.
Policard, A. (1950). *Wiss. ForschBer.*, **60**, 262.
—— and Collet, A. (1951). *C.R. Acad. Sci., Paris*, **233**, 1159.

Brit. J. industr. Med., 1952, **9**, 112.

A SAMPLING PROCEDURE FOR MEASURING INDUSTRIAL DUST EXPOSURE

BY

P. D. OLDHAM and S. A. ROACH

From the Pneumoconiosis Research Unit of the Medical Research Council, Llandough Hospital, near Cardiff

(RECEIVED FOR PUBLICATION JANUARY 15, 1952)

In the investigation of pneumoconiosis among workmen exposed to a dust hazard it is necessary to relate the development and progression of the disease over a number of years to the amount of dust in the air which the men breathe. In particular, it is necessary to relate the medical findings to the total dose of dust received by the workman during his time of exposure, for the effect of a given dust on the lungs must depend on the total amount inhaled and retained. It is our purpose in this paper to discuss the problems that arise in measuring the dose of dust, and to suggest a simple method by which they may be overcome. We shall deal specifically with dust sampling in coal mines, but we believe the same principles will hold in other industries.

The difficulties that arise when dust sampling results are to be related to the incidence of pulmonary disease in a defined population are of two distinct kinds. The one results from the variability of the concentration of airborne dust in the environment, the other from the necessity of turning measured concentrations into measures of dosage which may fairly be compared with indices of disease. There has been a tendency in the past to ignore both these sources of difficulty. Bloomfield (1933) and Dallavalle (1940) point out the necessity of estimating the mean duration of exposure to each type of dusty work, and of weighting the average dust concentrations estimated for each type by these times. However, they used measurements of dust concentrations made at fixed positions assumed to be typical of all working places, and variation in the duration of exposure from man to man was ignored. Other discussions of dust sampling in coal mines (Briscoe, Holt, Spoor, Matthews, and Sanderson, 1943); Monmouthshire and South Wales Coal Owners' Association, 1942, 1943, 1944; National Coal Board, 1949) have been concerned solely with methods of measuring the concentrations of dust, which, it was assumed, would be directly related to the production of disease without considering the duration of exposure of the men at risk. Bedford and Warner (1943), after a discussion of the difficulties involved in making time studies of the activities of coal face workers, also decided to rely on estimates of the mean level of dustiness. Evidence is given later which suggests that none of these assumptions may safely be made, and the results of attempts to correlate dust concentrations and indices of disease suggest that it is essential to adopt methods which make such assumptions unnecessary and which allow for the difficulties of varying concentrations and times at risk.

It will be seen that the total dose received by one man is given by the product of the mean concentration of respirable dust in each unit volume of air in the environment, the volume of air breathed in each unit of time, and the total duration of exposure. We are forced in practice to assume that the volume of air breathed, on the average, is the same from one group of men to another. The remaining factors will need to be estimated, and it will be necessary to study them in relation to those men, and only those men, who are included in the group studied by the medical survey. We do not need to know the dust concentration at a place where no one works, nor the duration of exposure of a man who is not in the group to which the medical findings apply. Moreover, it is essential that the sampling instrument used should give a measure of the concentration of that part of the dust which can be inhaled and retained by the human lung. The thermal precipitator, which is used in the examples given in this paper, can be made to give a measure of the respirable dust provided only those particles are counted which fall in a limited range of size. Any other type of sampling instrument with the required characteristics could, of course, have been used.

The data required are then the men at risk, the mean concentration of respirable dust in the working places of the men at risk, and the time during which the men are at risk.

The Men at Risk

In general, the medical survey can only usefully concern itself with selected groups of men in which the incidence or progression of pneumoconiosis may fairly be attributed to the dust conditions which are found (Hart and Aslett, 1942 ; Cochrane, 1951). Men will have to be excluded if they have a history of possible dust exposure elsewhere, or of exposure to dust other than that on the coal faces where sampling is carried out (in the screening plant on the surface, for example). It follows that it would be insufficient to estimate the average dose of dust inhaled by all the men in the pit and to compare it with the medical index of prevalence or progression amongst only the selected men. We must confine the dust sampling to places in the mine which are occupied by the men among whom the incidence of disease is to be measured. Similarly, we must establish the duration of exposure of these men and of no others.

The Mean Concentration

The variability of dust concentrations at one point on a coal face is such that within a single shift samples may give dust concentrations varying from near zero to a level two-and-a-half times the mean, even though each sample represents an average of the momentary concentrations occurring during a period of two to 10 minutes. Further, from one shift to another the mean level of dust concentration varies as much as the concentration within a shift. Attempts have been made to relate this variation to observable changes in the environment (ventilation, number of colliers, output, etc.) but without consistent success. It seems likely that it will always be necessary to sample during a substantial number of shifts. It has been found that, taking as many samples as possible during eight to 10 shifts (50 to 100 samples in all), the mean of a characteristic set of results may have a standard error of as much as 25%. The consequences of this are twofold ; the significance of quite large apparent differences in concentration may be left uncertain or, more seriously, the significance may be exaggerated by the failure of the sampling technique to detect all the sources of variation which affect the precision of the mean.

In addition to the variation of concentrations at one place with time, differences also occur from place to place, in particular from one end to another of a coal face. A technique of sampling which has been widely used is to sample at a single point at the return end of the face, or in the return roadway (Monmouthshire and South Wales Coal Owners Association, 1944 ; National Coal Board, 1949 ; Watson, 1949). It would be expected that concentrations near the air-intake end of the face would be lower than those at the return end, and indeed, in certain exceptionally stable conditions, a steady trend of concentrations along the face has been found which could be related to the number of working places or stints between the sampling point and the intake end. In general, however, conditions are too variable for such a trend to be established, and the possibility of a general law being found which would relate the concentration at any point to that measured at a fixed point seems remote. Thus the " return end " sampling procedure is inadequate for estimating the mean concentration to which all the colliers are exposed, and it follows that on each coal face a complete programme of sampling must be undertaken at each collier's working place, or at a sample of them sufficient to establish a precise mean. An illustration of the way in which return-end sampling may lead to misleading results is given later in this paper.

The Time at Risk

Coal-mining is not carried on with the routine regularity of many industrial occupations. The total length of shift, from the time the collier leaves the pit bottom to the time he returns to it, is usually fixed, but the duration of exposure to dust concentrations is as variable as the concentrations themselves. The time taken to reach the face from the pit bottom may be 10 minutes or 50 ; the time taken to get the agreed quantity of coal depends on the practical difficulties encountered from day to day, such as conveyor breakdowns and adverse roof conditions, and the mid-shift break may last anything from 10 to 40 or more minutes. When work ceases on the face, dust concentrations fall very rapidly to insignificant levels, so that periods of temporary stoppage may have to be considered as outside the time of dust exposure.

What allowance is made for these factors must depend on the way samples are taken during a shift. If a continuous sampling instrument were available, many of these difficulties would not arise. At present, the only instruments which are in use sample intermittently. If samples are taken with such an instrument only during " typical " conditions of dust production, then a complete time study of activities on the face must be undertaken so that the period over which the samples are valid may be known. This will involve a series of records as extensive as those of the dust samples themselves.

Alternatively, the samples may be taken at regular intervals throughout the shift or at times chosen previously by a random method. In this case details will still be required of the total time spent on the face, and, in addition, of the duration of the mid-shift break. This will be necessary because samples falling within the break must be treated separately from the rest, for several reasons, among which are difficulties of analysis caused by a number of exceptionally low concentrations.

A particular difficulty is the definition of the exact moment when the shift begins. This arises even if a continuous sampler is used. During the first quarter of an hour men will be arriving, taking up their places, and starting work, and dust concentrations may be abnormal. It is difficult to decide whether sampling should begin when the first man reaches the face, when the last man begins work, or at some intermediate moment. It might be thought that this difficulty is trivial, but these 15 minutes of exceptional conditions (and a corresponding period at the end of the shift) may form a substantial proportion of the total time of dust exposure, which is seldom more than 350 minutes, and has been found in one exceptional mine to be as little as 120 minutes.

Absenteeism is another source of variation in the duration of exposure, and it may also lead to changes in the dust concentrations to which men remaining at work are exposed. With fewer men at work on a face dust concentrations would be expected to be lower than usual. Substitutes are usually brought in to fill the places of the missing men, however. On occasions so many men may be absent that it becomes necessary to close one face for a shift and to transfer the remaining men to another face. Concentrations on the working face may then be higher than usual. In such circumstances it is difficult to decide what weight any samples taken should carry in determining the mean dose.

In practice a complete programme of time study of the collier's working life would be necessary if proper use were to be made of the results of dust sampling by present methods. Moreover, the effort put into dust sampling would not be used as efficiently as it might be unless the results of the time studies were available beforehand to suggest the proper distribution of the work. Obviously, concentrations to which a man is exposed for short periods only need not be measured with the same accuracy as those to which he is frequently exposed.

Practical Illustrations of Conventional Methods

We have suggested a number of difficulties which may be encountered in the course of an attempt to correlate dust and disease. It could be argued that in practice such difficulties are of small importance, and that the conventional methods of dust sampling are perfectly adequate.

A sampling method which is commonly used at present consists of taking return-end samples at different faces and on different shifts. The results are averaged, and the resulting figure is taken to represent the dust hazard to which the colliers are exposed.

Let us suppose that at two collieries, A and B, return-end dust samples have been collected. It is found that the mean concentration at colliery A is 3,000 particles/ml., and at colliery B, 1,000 particles/ml. Before it can be stated that the dust hazard at colliery A is three times that at B it must first be established beyond reasonable doubt that the men at colliery A have in fact been exposed to three times as much dust as those at colliery B.

The collieries each have two faces. At colliery A face A.1 has full dust suppression and a return-end concentration of 1,000 particles/ml., while face A.2 is a development face without dust suppression, and the return-end concentration is 5,000 particles/ml. Face A.1 is 200 yards long and face A.2 is 50 yards long. At colliery B both faces are equal in length, have full dust suppression, and each has a concentration of 1,000 particles/ml. It is clear that at colliery A the majority of the men are on face A.1 and are exposed to the lower average concentration. There are usually 50 colliers on A.1 and only 10 men on A.2. Assuming for the moment that return-ends are representative of the whole face then a better estimate of the mean concentration to which the 60 colliers are exposed is :

$$\frac{(50\times 1{,}000)+(10\times 5{,}000)}{60}=1{,}667 \text{ particles/ml.}$$

and not 3,000 particles/ml., the unweighted mean of the two return-end concentrations.

The respirable fraction of coal dust is nearly all less than 5 microns in diameter. Dust of this size settles out of the air very slowly. At colliery A the loader points, which produce a lot of dust, are in the return airways so that only the dust produced by the colliers themselves is included in the air they breathe. Consequently the first collier from the intake is exposed to very little dust and the dust from subsequent colliers builds up the concentration continuously to the return-end. The average concentration for such a face is thus about half that at the return-end. At colliery B, however, the loader points are in the intake and are very close to the face. A considerable quantity of fine dust is carried from them on to the face and in such a case the return-end may be no more dusty than the average for the whole face. We may conclude therefore that whereas the mean concentration to which the colliers at colliery A are exposed is 1,667/2 or 833 particles/ml., at colliery B the appropriate mean concentration remains at 1,000 particles/ml.

During the two surveys time studies were pursued at the two collieries during the sampling. At colliery A the coal faces are a long way from the shafts but the coal is very easy to get. The total time spent working in dust is just three hours. At colliery B, however, the coal faces are near the shafts and, the coal being very

hard to get, the men are working in dust for the complete seven-and-a-half hours of the shift, or two-and-a-half times as long as the men at colliery A. The total exposure to dust at A is thus $833 \times 3 = 2{,}500$ particle-hours per shift, and at B is $1{,}000 \times 7\frac{1}{2} = 7{,}500$ particle-hours per shift. Consequently there is at colliery A about one-third the amount of dust available for inhalation as there is at colliery B, although the simple averaging of return-end samples led to an exactly opposite conclusion. It is clear that if such dramatic changes can be produced, admittedly in an extreme case, by making allowance for the place of work and duration of exposure of the men at risk, then these factors must be estimated with as much care as the dust concentrations themselves. The rough estimates used in this simplified illustrative example would be inadequate.

Apart from these long-term considerations others arise in actually carrying out a return-end survey, such as the following, which have been encountered in recent surveys by the Pneumoconiosis Research Unit :—(1) Towards the latter part of the shift most of the men at the face had finished their work and left, while a few continued in order to complete their stints. As the men drifted out, so the concentration slowly dropped. When should sampling have ceased? (2) Because of difficulties the day before, the cycle of operations on a face had fallen behind and colliers from elsewhere in the pit were brought in to bring the strength up to double and so rectify the situation. Consequently, dust concentrations were very high and twice as many men were exposed to them. What weight should be given to this day's sampling, in view of the fact that the relative frequency of such exceptional days is not known? (3) A face consisted of a left-hand intake, a leakage return through which half the air passed, and a right-hand return which carried the remainder. Are there two sampling positions on this face? (4) There was a squeeze on the face the night before and the men worked on the roof during the coal-getting shift to open out the workings. What weight should be given to this day's sampling? (5) One seam was working longwall, and the other hand-got pillar and stall. In the pillar and stall workings the ventilation was very sluggish (air velocity less than 30 ft./min.) and the air filtered through many holes and old workings on its way through the seam. The men were working in pairs, scattered over the area. Where should the sampling position be located? (6) New entrants are put on the training face for a short period before proceeding to the main production faces. What weight should be given to samples taken here? (7) A fall of roof occurred in the main gate over a length of 10 yards. The colliers were put on cleaning the rubbish into trams. Should samples be taken and if so where and what weight should be given to them? It should be remembered that some of these decisions must be taken on the spot by the sampling technicians, and that others may cause considerable difficulty when the results come to be analysed.

The "Random Colliers" Method

The method of sampling we propose, to avoid the difficulties we have illustrated, is simple and economical. This method, which has been called "random colliers", consists of using the movements of individual colliers in the group studied by the medical team to determine the place and duration of dust sampling. Colliers are chosen by a random principle from this group, and, if an intermittent sampling instrument is being used, samples are then taken at the working places of each man at moments also chosen at random. The number of colliers followed in this way, and the number of samples taken in each working place, is determined by the resources of the sampling team and the fact that for the highest efficiency the maximum number of colliers should be followed. Since, however, practical considerations limit the amount of moving about done by the sampler, the best method appears to require each collier to be followed for one whole shift. Given the number of days which can be spent on the survey, the number of sampling instruments available, and the number of samples which can conveniently be evaluated, the number of colliers chosen and the number of samples per shift are deduced. The method is thus an extension of that proposed by Patterson (1939), in which a dust sampler follows an individual workman throughout his working day. It was clearly implied, though not stated, that this man is taken as typical, i.e. as a random sample of all those to be studied.

The time taken to prepare a complete sampling scheme of this type is no more than an hour or two, and the procedure is straightforward. The sampling itself is free from problems arising out of exceptional conditions, since the chosen collier's exposure provides a constant guide to the sampling procedure.

If automatic, continuous sampling instruments were available each random collier would take one with him from the pit top to his place of work and back again, and the instrument would sample continuously throughout his dust exposure. A satisfactory instrument of this kind must clearly be as portable and convenient as, for example, a Davy lamp.

It must be shown how this simple technique meets the objections and difficulties we have discussed. First, although nothing can eliminate the sampling errors introduced by the high variability of concentrations, it has been pointed out that the most efficient sampling method gives each momentary

state of the environment a weight proportional to its importance to the collier's health. This requirement is fully met by the proposed system, for the probability that a particular concentration is sampled is proportional to the time it endures.

The second difficulty, that of relating concentrations and their duration to obtain the total dose, is also overcome. The problem of deciding when to begin and end sampling, or when to begin and end the period during which samples are to be taken at random times, can be dealt with simply by deciding when the random collier enters and leaves the dusty environment. In principle we are sampling the dust exposure of the random collier throughout his whole day ; it is merely fortunate that his exposure begins and ends with his working hours and we are able to concentrate our attention on that period alone. We record its duration, and are then in a position to evaluate the total dose over whatever period is convenient.

Problems raised by absenteeism are similarly overcome. If the chosen man is absent his exposure is nil and no samples are taken. Providing no substitute is sampled instead, no bias is caused by a redistribution of the remaining colliers to fill his place. The unusual conditions so produced have exactly their due chance of being sampled since they may occur when the chosen collier is present and others are absent.

It might be thought that greater accuracy would be attainable if it were arranged that at least one collier from each face was always chosen. This would be a practical method if the number of men working on a face were always the same. In fact this is not so, and the gain in precision which would result from such "stratified" sampling is offset by the increased difficulty of combining the results and making the necessary records. It seems advisable on the whole to adhere to the purely random method we have described in which individual samples are combined in a simple manner, and no problem of differential weighting arises.

The details of the method are most easily shown by means of an example of its use in a survey at a South Wales colliery. Very few complications had to be added in practice to the simple scheme described above.

Practical Example of the "Random Colliers" Method

The men to be studied in this survey were the coal-getters, the colliers actually working on the coal face with their assistants, and the object of the survey was to estimate their exposure to respirable dust of whatever kind. A list of these men was provided by the colliery manager ; the total number was 142.

The time to be spent on the survey was limited to two weeks, or 11 coal-getting shifts (the pit was working alternate Saturdays). Four observers were available, and the sampling instrument to be used was the thermal precipitator.

Since this was to be in some respects an exploratory survey, it was decided to arrange the sampling so that an estimate could be made of the variability of concentrations experienced by an individual from day to day. For this purpose each collier was to be observed on two different shifts. The sampling scheme then depended on distributing the 44 shifts which could be sampled by four observers on 11 working days among 22 randomly chosen colliers. This was done by taking from a table of random numbers the first 22 which were less than 143. The chosen colliers were then those in the list numbered from 1 to 142 whose serial numbers had been selected. By a further use of random numbers a table was drawn up in which four different colliers (one to each observer) were allocated to each of the 11 sampling days, in such a way that each collier appeared on two different days.

The number of thermal precipitator samples that can be taken during a survey is limited by the labour involved in counting the deposits. It was decided in this case that not more than about 450 samples should be taken. Thus about 10 samples were to be taken each shift by each observer, and the maximum was set at 12. The total period of exposure during a shift cannot be predicted with any accuracy, but, in practice, work on the face does not begin before 7.45 a.m. nor end after 2.15 p.m. It could be assumed that at this colliery there was no material dust exposure while travelling from the shaft to the face. To ensure a random distribution of samples throughout the shift, recourse was had again to random numbers, and for each of the 44 shifts a list of 12 times was prepared, lying between 7.45 a.m. and 2.15 p.m. and separated by at least eight minutes (each sample took three minutes, and five minutes was allowed for adjustment of the instrument between samples).

Each observer's daily programme was thus as follows. He met his allocated collier at the lamp-room on the surface or at the deputy's cabin underground, and travelled with him to his place of work. The thermal precipitator was set up, upwind of the collier and at head level, as close to him as could be managed without interfering with his work. The distance was usually about six feet. The first sample was taken at the first time on the prepared list which was after the time of arrival. Successive samples followed until the collier left the face. The time of arrival and departure was noted, and the time and duration of the mid-shift break.

Results

Between the time the sampling scheme was prepared and the beginning of the survey one collier left the pit. Unfortunately he was one of the selected men, and in consequence the sample consisted of 21 men chosen out of 141. One collier was an absentee on both of the days he was due to be observed, and another five were absent on one of their days. Some of the samples taken were lost by contamination, but it has been observed

that such losses are not correlated with the level of the dust concentrations, so that the results are not biassed by the loss of some of the data. The full results are given in the appendix table.

The mean concentration to be estimated from the data is that to which the average collier was exposed. The mean concentration to which one collier was exposed will be given by the unweighted mean of the means for each of the days for which he was observed. The final figure will be the mean, again unweighted, of these. This was found to be 716 particles per ml. In this calculation the readings obtained during the mid-shift break, which were all effectively zero, were excluded. We therefore calculated the durations of exposure from the recorded shift lengths, excluding the length of the mid-shift break. These were found to be uncorrelated with the concentrations observed. Thus the mean duration of exposure was taken as the grand mean of all the observed shift lengths less break times, and the figure that resulted was 311 minutes. This, multiplied by the mean concentration, gives 222,676 particle-minutes per ml., or 3,710 particle-hours per ml. Assuming a constant volume of air breathed per minute, and no dust exposure outside working hours, we may conveniently express this figure as proportional to a dose of 3,710 particles each working day.

An analysis of variance was carried out on the data, for which purpose it was necessary to work with the logarithms of the measured concentrations and shift lengths. It was found that significant variation was occurring in the dust concentrations from one collier's experience to another's, and from one day to another in the same collier's experience. Estimates were made of the magnitude of this variation, and of the variation of successive samples taken on the same day, and finally 95% confidence limits were calculated for the dose given above. These were 3140 and 4080.

The greatest practical difficulties found in carrying out the survey came from the use of a short-period sampling instrument. An automatic continuous sampler, operating from the moment the collier entered the mine to the moment he left it, would make it unnecessary to make special provision for the effect of mid-shift break and the irregular shift length. No difficulty was experienced in explaining the idea of "a cross-section of the pit" to the individual colliers chosen, nor did any collier show resentment at being picked out. In fact the individual's usual reaction was one of interest and cooperation, just as has been found by those engaged in carrying out public opinion polls or sample surveys of sickness. There was no sign that he modified his habits of work because of the presence of the sampling instrument, and it should be remarked that even if he did his exposure would be substantially unaltered; the collier does not breathe his own dust to any material extent, but rather that produced upwind of him by his workmates.

Long-term Surveys

In conclusion we must refer briefly to the procedure to be adopted in continuing a survey for a long period, or in carrying out a series of short surveys at regular intervals, as will generally be essential in correlating dust exposure with disease. The chief difficulty to be considered is the possibility that the population of the pit may change, some of the original men leaving and new men taking their places. The difficulty may be met by ensuring that the population sampled remains, throughout, the population in which incidence of disease is to be studied.

Example of a Long-term Survey System

One system which would satisfy this requirement may be illustrated by an example. Suppose a colliery which is to be subject to periodical radiographic surveys has initially 200 coal-getters on its books. Each year some of these will leave the colliery or move to jobs away from the face and others will take their places. To the original list of 200 men a number of blanks are added, perhaps 40 if the population of colliers is known to fluctuate in numbers by about 20%. These 240 numbers are put into random order. Then, if 10 sampling instruments are available, the first 10 men on the randomized list form the first random colliers. As men leave their numbers become vacant, and as new men join their names are placed against the first blank spaces on the original list and against the corresponding numbers on the randomized list. In this way sampling is continued, taking always the next 10 occupied numbers on the randomized list. When the end of the list is reached the procedure begins again at the beginning. It will be seen that in this way the probability that a man will be sampled remains, as is necessary, proportional to his time at risk. The method can obviously be extended to cover a series of surveys as well as a continuous one. In general, provided it is possible to deduce a valid measure of disease incidence or progression, a valid measure of dose of dust will be calculable by following a strictly comparable procedure.

Discussion

In this paper we have attempted to show that a proper evaluation of an industrial dust exposure must take into account variations in the concentration of the dust and of the duration of exposure of the men at risk, who must be the same men upon whom the incidence of pneumoconiosis is observed. This must be so whether it is the dose of all respirable dust or of some particular part of the respirable dust (the free silica, for example)

which is to be compared with the incidence of disease.

Methods of sampling that have been employed in the past mostly rest upon the assumption that in the long run the average of a number of dust samples taken on any principle will lead to a quantity which can validly be compared with an observed disease incidence. We have suggested that even if such assumptions are justified, and we have presented some evidence to the contrary, these methods cannot be economical. The concentrations measured by a sampling instrument do not all carry equal weight as measures of the dust hazard; their individual importance must depend on their duration and on the number of workmen exposed to them. This criticism applies even more strongly when care is taken to evaluate durations of exposure by a separate and extensive series of time studies. A random collier survey involving an equal amount of dust sampling makes the most economical use of the labour and time spent, for it automatically gives to each concentration a weight proportional to its importance in producing pneumoconiosis. We may remember here the remark of Belt and Ferris (1942): "The collier's lung is, in a very real sense, his occupational log book; it retains a qualitative and an indelible record of the mineral particles breathed during life." By basing the programme of sampling on the collier's occupational log book, we are imitating with our sampling instrument the experience of his lungs.

Summary

Any method of estimating the dust exposure of workmen for correlation with the incidence of pneumoconiosis must satisfy the following criteria:—(1) The exposure estimated must be that of the same workmen upon whom the incidence of pneumoconiosis is to be found. (2) The duration of exposure must be measured as well as the concentration of dust. (3) Account must be taken of the effects of the variability of exposure, both in duration and intensity.

Evidence is produced showing the errors that can arise from ignoring these criteria, and a new method of dust sampling is described which satisfies them. This method is based on following with a sampling instrument throughout their working day individual workmen chosen by a random principle from all those on whom the incidence of disease is to be measured.

A practical example of the use of this method is given, and it is demonstrated that the method is simple to execute and makes the most economical use possible of a given amount of sampling.

We should like to thank our colleagues in the Pneumoconiosis Research Unit for helpful discussions, and the South-Western Division of the National Coal Board for the facilities so readily placed at our disposal.

References

Bedford, T., and Warner, C. G. (1943). *Spec. Rep. Ser. med. Res. Coun., Lond.*, No. 244, p. 1.

Belt, T. H., and Ferris, A. A. (1942). *Ibid.*, No. 243, p. 219.

Bloomfield, J. J. (1933). *Publ. Hlth Rep., Wash.*, **48**, 1343.

Briscoe, H. V. A., Holt, P. F., Spoor, N., Matthews, J. W., and Sanderson, P. M. (1943). *Spec. Rep. Ser. med. Res. Coun., Lond.*, No. 244, p. 79.

Cochrane, A. L. (1951). 'The Application of Scientific Methods to Industrial and Service Medicine', p. 97 (Proc. Conf. held 1950). H.M.S.O. London.

Dallavalle, J. M. (1940). *Publ. Hlth Bull., Wash.*, No. 250, appendix I.

Hart, P. d'A., and Aslett, E. A. (1942). *Spec. Rep. Ser. med. Res. Coun., Lond.*, No. 243, p. 1.

Monmouthshire and S. Wales Coal Owners' Ass., Coal Dust Research Committee Reports No. 7 (1942), 9 (1943), 11 (1943), 13 (1944).

National Coal Board (1949). Circular 'Employment of Pneumokoniosis Cases. The Sampling of Air-borne Dust for the Testing of "Approved Conditions".'

Patterson, H. S. (1939). *Bull. Insn. Min. Metall.*, No. 417.

Watson, H. H. (1949). Proc. 9th Int. Congr. industr. Med., London, 1948, p. 1030. Bristol.

APPENDIX

RESULTS OF "RANDOM COLLIERS" SURVEY

Collier	Total Time Spent on Coal-face (min.)	Duration of Mid-shift Break (min.)	Thermal Precipitator Samples (No. of particles per ml. between 0·5 and 5·0μ)
1	315	25	1,630 800 1,100 920 1,500 1,770 980 1,430 1,540
	430	15	950 <100*
2	—	—	*Collier no longer working at pit*
3	350	20	<100 1,320 650 <100 <100 360 (<100)† <100 220 180
	365	20	800 1,180 1,130 880 430 430 390 360 460 440
4	345	20	1,750 1,690 2,030 2,730 1,980 940 1,360 2,070 2,190 830 2,650
5	—	—	*Collier absent on both occasions*
	—	—	
6	270	20	570 <100 <100 (210) 310 740 2,000 520
	335	25	240 170 450 560 320 280 (<100) 320 520 820 520 510
7	325	40	390 700 920 1,240 1,580 1,530 920 530
	365	35	1,110 730 (<100) (<100) 750 1,090 720 1,130 580 770
8	300	25	580 770 470 (<100)
	395	25	350 1,460 800 1,250 1,150 1,010 (<100) 860 920 760 840
9	—	—	*Collier absent*
	375	25	670 550 480 520 530 450 1,490 950 790 560
10	375	20	230 410 510 460 490 290 230 270 150 1,060
	360	35	1,450 1,100 1,020 900 340 270 240 550 260 380 310
11	390	25	2,770 4,510 2,240 280 500 380 400 300
	380	20	460 480 750 510 (<100) 460 270 410 520 650 780 650
12	375	25	570 470 490 470 500 630 640 710 450 320 180
	380	25	690 1,460 1,150 1,030 980 350 440 560 2,290 810 710 1,330
13	‡	‡	770 650 870 1,280
	—	—	*Collier absent*
14	365	20	330 530 430 (<100) 640 620 550 680
	345	25	1,240 1,290 1,150 1,160 1,040 470 760 790 720 730 (<100) 1,800
15	380	20	550 <100 200 130 490 420
	—	—	*Collier absent*
16	390	25	180 460 590 550 370 350 270 290 620 290 250 230
	355	50	170 <100 480 480 690 (<100) (<100) (<100) (<100) 380 460 400
17	375	25	580 940 660 920 760 760 750 790 1,230 1,080 1,390 900
	355	20	1,630 1,470 1,560 1,790 1,780 1,250 1,000 840 1,000 1,870
18	325	25	*Samples contaminated and uncountable*
	375	30	920 620 1,010 1,040 980 (<100) (<100) 530 1,040 870 770
19	360	20	690 700 1,030 (<100) 530 610 510 1,060 260 710 370 <100
	330	25	570 800 710 (<100) (130) 420 530 620 520
20	340	15	<100 760 2,660 3,680 430 230 120
	—	—	*Collier absent*
21	345	30	1,090 660 540 1,120 840 620 820 710
	—	—	*Collier absent*
22	350	50	570 760 840 690 1,370 900 650 660
	380	20	490 370 650 830 670 640 440 680 380 400 610 400

* Samples too sparse to count are recorded thus.
† Samples in brackets were obtained during the mid-shift break.
‡ Not recorded.

Brit. J. industr. Med., 1952, **9**, 120.

DUST SAMPLING AND LUNG DISEASE*

BY

C. N. DAVIES

From the London School of Hygiene and Tropical Medicine

No instrument for sampling airborne dust or mist collects a sample which is truly representative of all the particles present. There are errors associated with the random variation of the cloud in time and position and instrumental errors due to the act of sucking the cloud into the sampling apparatus or to inherent failings of the dust-separating or measuring mechanism which cannot be entirely eliminated. Fine particles, coarse particles, or both, may be lacking in the final analysis, and the original particles may be changed during collection. In favourable circumstances a fair picture of the airborne dust can be built up by combining several methods of securing samples and using a variety of techniques for their evaluation.

The Principle of Selective Sampling

A laborious reconstruction of the original cloud is seldom necessary for gauging a risk to health. The danger due to a toxic gas is measured by its concentration and the same is true of a cloud of one substance dispersed in particles of uniform size. But it is usual for dust and mist to contain a range of sizes, only some of which need to be sampled accurately because of their greater significance. Unless dangerous particles happen to lie in the coarse end of the size distribution, correlation between health risk and mass of dust is likely to be poor and it will be necessary to investigate microscopically the particle size distribution in the cloud.

On the other hand, the mass of dust in a selected sample, containing only particles in the dangerous size range, may be a good measure of health risk. If the sampling instrument selects this range spontaneously, microscope work is avoided and analysis of the results is reduced to simple weighing. Because of the great simplification which is promised by selection, it is necessary as a preliminary to dust surveys to consider what constitutes the dangerous range of particle size.

The full benefit of selective sampling is gained when the dust is slowly absorbed, or reacts very slowly in the tissues of the body. Ingestion and contamination of the skin and eyes produce no significant pathological effects, and natural elimination from the bronchioles, bronchi, and upper respiratory tract of the larger particles is probably sufficiently rapid and complete to constitute an adequate defence of these regions. Harmful effects, therefore, can be ascribed fairly confidently to particles which are small enough to penetrate deeply into the lungs. Alumina-silica fume and vanadium pentoxide, silica and coal dusts, for example, fall into this category.

The action of reactive or soluble particles of dust or liquid is complicated by the possibilities of evaporation and chemical change in the atmosphere as well as by localized tissue sensitivity. It is impossible to generalize about such particulate clouds beyond saying that mass concentration of total airborne material is likely to be a better measure of health risk than it would be for dusts of the preceding group. This implies that the coarser particles, which contribute largely towards the weight, are likely to be dangerous, although it is by no means certain that symptoms will be confined to the upper respiratory tract, where the larger particles are filtered out, because systemic effects may arise. Conversely, proceeding to particles of molecular dimensions, some gases can cause upper respiratory symptoms due to their rapid absorption, while others produce lung reactions ; again, fine particles of tobacco smoke, of sizes which are almost entirely retained in the alveoli and fine bronchioles, are believed to have some association with cancer of the bronchi where their retention factor is very low. In general, it is not safe to assume a simple connexion between the size of particles responsible for lesions and the places where they occur.

It seems, therefore, that the benefits of selective sampling, in which an attempt is made to simplify the investigation of a dust hazard by restricting

* A summary of this paper was presented at the tenth International Congress of Industrial Medicine, Lisbon, September, 1951.

attention to a limited range of particle sizes known to be dangerous, are confined to fields in which the chemical action of the particles is slow; this restriction means that particles which are to be selected by the sampling device should be those which are retained in the human lung for long periods. Particles of very low solubility deposited in the upper respiratory tract, or on any part of the ciliated epithelium of the bronchial tree, will be excreted and can have no conceivable effect on a long term basis.

If the actual sample can be collected or assessed after the elimination of all particles, which would have been deposited during respiration elsewhere than in the alveoli, then the ideal selective sampler has been achieved and the material available for chemical and physical examination will be similar to that which remains in the lung. The need for studying functions of the size distribution would not be entirely removed by such a technique because the toxic effect per particle must depend on weight, surface area, or other factors; nevertheless, it is likely that a relationship between disease incidence and mass of dust in the restricted, "alveolar" sample would be shown.

Gaddum (1941) was probably the first to apply the principle of selective sampling by drawing air into an impinger through a bent glass tube. The coarser of the suspended particles were deposited in the curve of the tube, due to inertia, while the finer ones, being able to sweep round the bend, were collected in the impinger; the dimensions of the apparatus were chosen so that the tube retained particles of sizes which were filterable by the human nose and the impinger collected a "lung" sample. Before discussing sampling methods it is therefore necessary to consider the filtering action of the respiratory tract.

Retention of Dust in the Human Alveoli

Since the publication of the author's review of inhalation risk and particle size (Davies, 1949) several papers have appeared which give important additional information on the access of dust to the alveoli.

There are two distinct experimental methods available for studying this problem. An analysis can be made of air exhaled by living subjects, and its content of particles compared with the quantity inhaled. This technique has been adopted recently by Brown, Cook, Ney, and Hatch (1950), by Dennis and Sawyer (1950), and earlier by van Wijk and Patterson (1940). Alternatively, undissolved dust can be extracted from the lungs of deceased miners and rock workers and its size distribution determined and compared with the size distribution of airborne dust in their former work places. The latter method has been tried by Gessner, Rüttner, and Bühler (1949), by Bedford and Warner (1950), and by Cartwright and Nagelschmidt (1951).

Some of the particles which are deposited beyond the respiratory bronchioles, where there is no ciliated epithelium, will be taken up by phagocytes, or otherwise penetrate into the lymphatics and will remain available for subsequent extraction and estimation in the lung residue, if they are insoluble. Others, however, will be carried by phagocytes to the ciliated surfaces above the respiratory bronchioles and will be eliminated from lung tissue. The limits of particle size outside which phagocytosis is ineffective are unknown, but they must exist.

The two methods of investigation should yield different results for particles which are neither too large nor too small for effective phagocytosis; exhaled air analysis would record that a greater proportion of these sizes was retained in the lung than did the analysis of lung residues from which material would have been lost by phagocytosis to the ciliated epithelium and possibly by solution.

Particles are deposited in the upper bronchial tubes by impingement, with a gradual transition to sedimentation in the finest bronchioles and alveoli. Since the effectiveness of both these processes is measured by the value of ρd^2 for particles of similar shape, ρ being the density and d the diameter of the particle, it follows that experiments performed with particles having density ρ can be expressed in terms of the diameters of particles of unit density by multiplying the actual diameter by $\sqrt{\rho}$, subject to certain limitations of the breathing pattern which are discussed below. In this paper all the results quoted have been rendered comparable by adjusting the particles sizes to unit density.

The retention of particles between 0·2μ and 1·5μ diameter takes place almost entirely in the alveoli and respiratory bronchioles; this will be termed alveolar retention. Larger particles are distributed throughout the bronchial tree, though it becomes more and more probable with increasing size that they will precipitate before reaching the alveoli.

Brown, Cook, Ney, and Hatch measured the carbon dioxide concentration in expired air simultaneously with their particle analysis; by assuming that the exchange of fine particles between alveolar and tidal air was similar to the exchange of gas, they were able to distinguish alveolar retention of particles. Their curve for maximum alveolar retention has been reduced for density, as explained above, and is plotted on Fig. 1, and the corresponding data for particles retained above the alveoli are shown in Fig. 2. In Fig. 1 the results of van Wijk and Patterson and of Dennis and Sawyer have

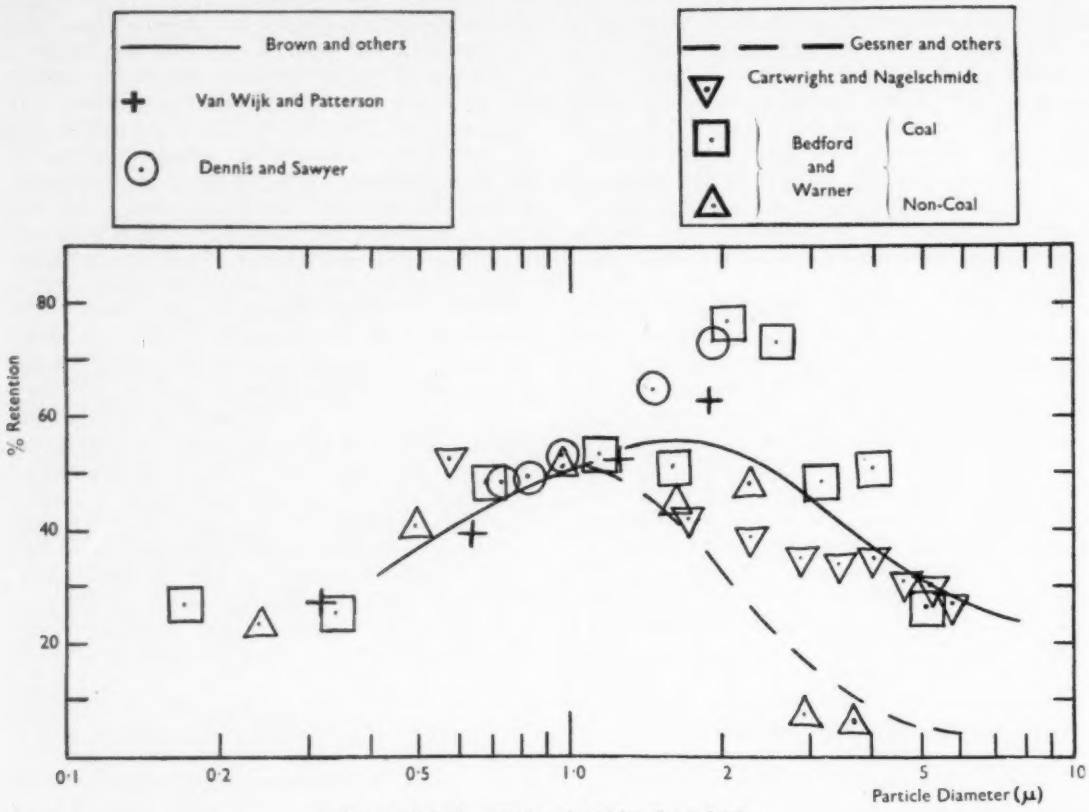


FIG. 1.—Alveolar retention of particles of unit density.

also been plotted, using only reduced particle diameters which are less than 2μ. These points fall quite close to the curve of Brown and others, though the points at 1·9μ are high, presumably because some particles are included in the alveolar retention which had actually been deposited above the respiratory bronchioles.

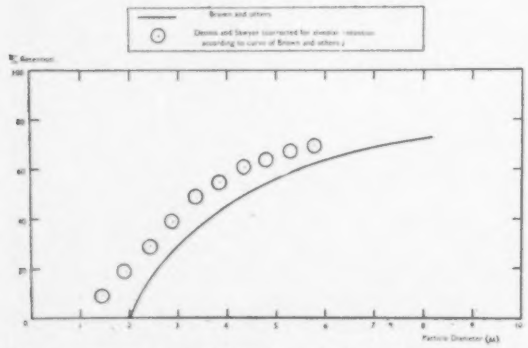


FIG. 2.—Percentage of inhaled dust which is retained above the respiratory bronchioles (unit density).

If the data of Dennis and Sawyer, corrected for alveolar retention according to Brown and others, are plotted it will be seen in Fig. 2 that all the points lie above Brown's curve, though not by a large amount. To bring the points on to the curve it would be necessary to have a greater proportion of alveolar retention than is given by Brown's maximum curve. It should be pointed out that all these workers took averages from a number of separate experiments. Dennis and Sawyer used 25 subjects, Brown and others made 100 tests, and van Wijk and Patterson did 40 experiments.

From their analyses of lung residues, Gessner, Rüttner, and Bühler computed a dust retention curve by assuming that particles between 0·5 and 0·75μ were completely retained. This is not true, even allowing for the high density of their dust, and their retention curve has been adjusted in the light of the air analysis experiments described above so as to make the alveolar retention 52% at 1μ diameter with unit density. The resulting curve is plotted on Fig. 1. This adjustment assumes that both solution of particles and their transport by phagocytes to the

bronchioles are either negligible or independent of particle size over the range investigated. The relationship between air analysis and lung residue experiments is unaffected by phagocytosis to the lymphatic system.

Bedford and Warner, examining lung sections microscopically, separated the particles into coal and non-coal by their appearance, and gave distributions of dust in lung nodules and also in reticulation areas. Averages of the latter have been used as the differences are not significant. Figures from their Table VI have been extracted, and lung dust compared with airborne dust after firing ripping shots for the non-coal particles, and after shots in coal, for the coal dust. Adjustment of lung retention has been made to 52% alveolar retention at 1μ, for particles of unit density, supposing the density of the fine coal particles to be 1·3 g./cc. and the non-coal to be 2·6 g./cc. These points are plotted on Fig. 1. The non-coal particles give points which lie in very good accord with the three air analysis experiments below 1μ and then follow Gessner's curve quite closely up to 4μ. The coal particles also give points which agree with all the other results below 1μ, but above 1μ alveolar retention is much higher than it was for non-coal ; it is apparently above the mean curve of Brown and others in the region of 2μ but it is quite close to their curve for larger particles.

The points on Fig. 1 from the work of Cartwright and Nagelschmidt (1951) are taken from Fig. 8 of their paper, and relate to a comparison of trypsin digested lung residues with the mean of three airborne coal dusts from South Wales, not, however, from the actual pits in which the men had worked. Particles were counted and sized under the microscope. Reduction to unit density and 52% retention at 1μ have been carried out as before. These points agree very well with the curve of Brown and his colleagues, from 4μ to 5μ diameter, and though they show a lower retention between 2μ and 4μ, the values are well above the curve of Gessner, Rüttner, and Bühler and the non-coal points of Bedford and Warner.

It is clear that on the basis of the adjustment of lung residue size distribution to 52% alveolar retention at 1μ (unit density) a very good measure of agreement between different laboratories and techniques has been obtained regarding alveolar retention of particles below 1μ diameter, irrespective of the nature of the dust, which rather argues against the possibility that solution was appreciable in the lungs concerned. This in turn suggests that phagocytosis of these very fine particles upwards beyond the respiratory bronchioles is not an important factor. Perhaps they are too small for the phagocyte to sense, or for the probability of chance encounters to be significant, or even to stimulate the production of phagocytes.

Above 1μ in Fig. 1 implies that clay and quartz particles are retained in the alveoli less abundantly than would be expected from air analysis. This might be explained by the phagocytosis of some of them to the ciliated epithelium. On the other hand, coal dust seems to be retained very much as the experiments of Brown and others indicate, about whose curve the points are fairly evenly spread. Such a difference between coal and quartz, if substantiated, might be accounted for on the hypothesis that coal particles were less effectively transported by phagocytes.

Further analysis of lung residues, and a more accurate correlation with the working environment, is necessary before such ideas can be accepted, particularly in view of the very difficult particle size analysis involved and the uncertainty attached to the separation of lung residues without disturbing the particle size distribution. All the same, it is fair to comment on the remarkable agreement below 1μ and near 5μ because such widely different experimental techniques were employed.

Dependence of Particle Retention on Breathing Rate

We must also consider the possible effects of variation in breathing pattern. For example, in normal respiration, the tidal air is 0·5 litres and the functional residual air is 2·9 litres (Davies, 1949) so that, if all the alveoli worked together, only 15% of the air in an alveolus would be expelled. For maximum respiration, the figures are 3·8 and 1·6 litres, so 70% of the volume of an alveolus is used.

Now alveolar retention depends on the number of particles which reach those regions ; this, in turn, is a direct function of the volume of tidal air, T. It is also related to the time they stay there which is an inverse function of respiration frequency, R. Hence, alveolar deposition is measured by the ratio T/R. During heavy work both T and R increase, but the ratio does not change very much. In contrast, impingement of larger particles is favoured by a high value of T and a high air velocity, which is a direct function of the respiratory frequency. Thus, the deposition of particles in the upper lung, due to impingement, is favoured by increasing the product TR, or minute volume, which, of course, increases rapidly when the body demands more oxygen.

As can be seen from Table 1, the values of 100T/R covered in the experiments we have been discussing do not vary a great deal, so it is reasonable to expect that the alveolar retention, at least, for sizes below 2μ, will be similar, and our comparison

of these experiments will not be invalidated by differences in the cycle of respiration.

Some idea of the dependence of retention on breathing pattern can be obtained from the theoretical calculations of Landahl (1950) and the experiments of Landahl, Tracewell, and Lassen (1951). Unfortunately, these experiments appear to be marred by an overestimate of particle size by a factor of 2, just as was the case in the earlier experiments of Landahl and Herrmann (1948). Attention was drawn to this descrepancy in the writer's previous paper (Davies, 1949). On Fig. 3 the mean curve of the experiments we have discussed in the last section is plotted. They were done with values of 100T/R between 3·2 and 4·7 (Table 1). The experimental points of Landahl and others, with 100T/R equal to 3 and 12, are shown joined together by a vertical line. Each of these lines falls too far to the right, but, if the particle size is reduced by one half, a curve through the mid-point of the joining lines lies very close to the mean curve of alveolar retention, which it cuts. The minima of the curves occur at the same particle size, while the only marked deviation in trend is above 1·5μ diameter where upper lung retention has become appreciable.

This defect does not prevent the use of Landahl's

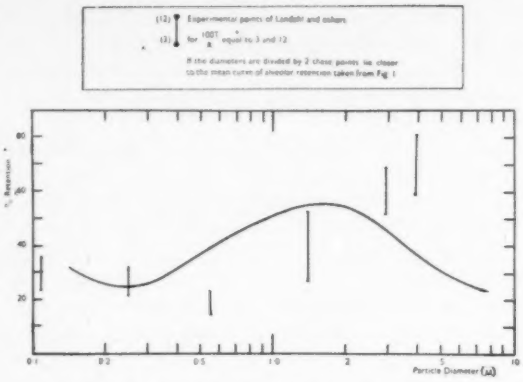


FIG. 3.—Alveolar retention of particles of unit density.

work to demonstrate the effect of breathing pattern. In Table 2 it is shown that alveolar retention is a direct function of 100T/R for particles below 1·9μ diameter (on Landahl's figure, 3·8μ) and that the dependence is less for larger particles owing to impingement in the upper bronchial tubes coming into play. Table 3 shows that upper lung retention is almost unchanged as long as the minute volume, TR, is held constant, although wide individual ranges of T and R are covered.

TABLE 1

PATTERN OF RESPIRATION

Experiments	Respiration Frequency (R) (min.)	Tidal Air (T) (litres)	100T/R. (l./min.)	Minute Volume (TR) (l./min.)
Brown and others	15	0·7	4·7	10·5
van Wijk and Patterson	19	0·9	4·7	17
Dennis and Sawyer	15·7	0·68	4·3	10·7
Mining	25	0·8	3·2	20
Hard exercise	30	1·3	4·3	40

TABLE 2

RETENTION OF PARTICLES IN THE LUNG ACCORDING TO THE EXPERIMENTS OF LANDAHL, TRACEWELL, AND LASSEN (1951)

Respiration Frequency (R) (min.)	Tidal Air (T) (litres)	100 T/R	Percentage Retention of Particles of Diameter (μ)*						
			0·11	0·25	0·55	1·4	2·9	3·8	6·3
15	0·45	3	24	22	15	27	52	59	86
7·5	0·9	12	36	32	23	53	69	82	93
5	1·35	27	46	41	32·5	65	81	89	96

* Particle diameters are quoted as published but are probably too large by a factor of 2.

TABLE 3

RETENTION OF PARTICLES ABOVE THE RESPIRATORY BRONCHIOLES AS CALCULATED BY LANDAHL AND OTHERS

Respiration Frequency (R.) (per min.)	Tidal Air (T) (litres)	TR	Percentage Retention of Particles of Diameter (μ)*				
			0·25	0·6	2	6	20
15	0·45	6·75	2	2	5	42	99
7·5	0·9	6·75	2	2	9	49	97
5	1·35	6·75	3	1	9	50	93

* Particle diameters quoted as published.

Design of a Selective Sampling Apparatus

Having reviewed the best information available on the size-selecting properties of the human lung, it is appropriate to consider the retention curve to which a dust sampler ought to be designed in order that it should sample alveolar dust and be suitable for assessing long term health risks due to particles which dissolve in the lung fluids only slowly, if at all.

It is probably safer to neglect the ascent of particles to the bronchioles by phagocytosis since correction for this would be entirely speculative. To obtain strict correlation between dust samples and incidence of disease, the acceptance curve of the sampling instrument should be biased by a toxicity factor ; existing knowledge, however, will only permit this to be done in exceptional cases so it will not be attempted in this paper.

Maximum alveolar retention occurs between 1·5μ and 2μ diameter and amounts to 50 to 60%. It is simplest to design selective sampling instruments to accept all dust particles of this size and to grade the cut-off of the larger sizes to about 50% at 5μ, at which size alveolar retention is equal to about half of its maximum value.

Although there is a pronounced falling off of alveolar retention from 1μ down to 0·3μ diameter, there is no doubt that still smaller particles are retained with increasing efficiency due to Brownian motion. There seems to be no advantage in trying to make the selector device match the alveolar retention curve for small particles, which would be very difficult, as the contribution of the smaller sizes to the mass of the sample is inconsiderable and little is known of the relative toxicity. We therefore aim at sampling all particles which are smaller than 1·5μ to 2μ diameter, together with a decreasing proportion of the larger sizes, so that 50% of the 5μ ones are collected.

It has been shown by Walton (1950) that if size separation is performed by elutriation of the air before sampling the residual fine portion, then, of the particles of diameter d originally present in the air, the fraction collected in the sample is equal to $1 - (d/D)^2$, where D is the upper limit of size in the sample. If we decide on 50% acceptance at 5μ diameter for particles of unit density this formula gives D = 7·1μ and 92% acceptance at 2μ. The complete acceptance curve is plotted on Fig. 4 where it is compared with a curve computed from the mean alveolar retention function (Fig. 3) by adjusting the maximum to 100%. If anything, this

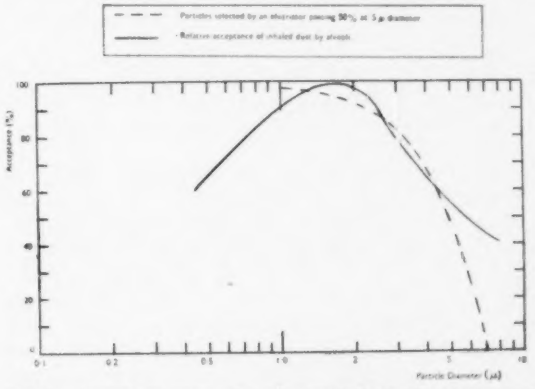


FIG. 4.—Acceptance curve of a selective dust sampling instrument with particles of unit density.

cut-off is rather too sharp, in comparison with alveolar retention, but it is unlikely that a practical instrument would exactly reproduce this theoretical formula and the appearance of a tail of coarser particles would be expected.

When the actual sampling instrument is preceded by a selector device operating to this specification, then the dust which it receives will be similar in size distribution to that reaching the alveoli of the human lungs. If the density of the particles is not equal to unity, which has been the basis of all our calculations, this conclusion is not modified in any way because segregation in the elutriator and deposition in the alveoli both depend on the same physical property of the dust particles, namely the rate of fall under gravity, and are altered in the same ratio. What does happen, of course, is that

the size of the particles accepted changes according to the law ρd^2 = constant. Change in size with density is shown for various values of the acceptance in Table 4. Similarly, the shape of the particles does not affect the relationship between the dust in the sample and that in the alveoli, except, perhaps, that extreme shapes like asbestos fibres may lodge more readily against the walls of the bronchial tubes.

TABLE 4

PERCENTAGE ACCEPTANCE VALUES FOR A SELECTIVE SAMPLER OPERATING BY ELUTRIATION AS A FUNCTION OF PARTICLE DENSITY

Percentage Acceptance	Diameter of particles (μ) when Density (g./cc.) is			
	1	1·5	2	2·5
10	2·2	1·8	1·6	1·4
20	3·2	2·6	2·2	2·0
30	3·9	3·2	2·7	2·5
40	4·5	3·7	3·2	2·8
50	5·0	4·1	3·5	3·2
60	5·5	4·5	3·9	3·5
70	5·9	4·8	4·2	3·7
80	6·3	5·2	4·5	4·0
90	6·7	5·5	4·7	4·2

An important point in connexion with the precipitation system, or method of measuring employed to assess the selected sample, is its sensitivity to very small particles. It was pointed out earlier on in this paper that it would be more rational to correlate the weight of selected samples of dust with the incidence of pneumoconiosis than total weight or surface area. All the same, it must be remembered that alveolar retention is large for submicroscopic particles, and although the mass of these in dust clouds is usually small, the numbers of particles may be considerable, as has been shown by the analyses of Wynn and Dawes (1951) and the electron microscope studies of McCartney (1945) and of Sharpe and Hounam (1950). Hatch (1950) has pointed out that there are no grounds for assuming that these very small particles have no significance in certain lung disorders.

Summary

Selective dust sampling, in which only fine particles are collected, often offers better prospects of correlation with the incidence of disease than the assessment of total concentration by weight, because the coarser particles are deposited in the higher bronchial tubes, from which they are excreted, while only small sizes reach the alveoli. The application of this principle is discussed in relation to the nature of the airborne dust.

With a view to defining a suitable acceptance curve for sampling apparatus, recent work on the retention of dust in the human alveoli is examined. For particles of unit density, the maximum alveolar retention is between 50% and 60% of the dust inhaled and occurs between 1·5μ and 2μ diameter; about 30% of the 5μ particles are retained.

It is possible that when coal dust is inhaled the proportion which is retained in the alveoli is greater than in the case of quartz or rock dust.

Minimum alveolar retention occurs at 0·25–0·3μ, smaller particles being more completely deposited.

Alveolar retention from 0·3μ to 2μ diameter depends on the ratio of tidal air to frequency of respiration and does not change rapidly with rate of working; upper lung retention varies with minute volume, however, and is therefore sensitive to muscular activity.

Selection of particles for sampling by an elutriator designed to accept 50% at 5μ diameter (unit density) should give a good approach to an alveolar sample. Variation of the density or shape of the particles will affect alveolar retention and elutriation in the same way, since both depend on the sedimentation rate of the particles, so that a single selector will give a constant performance with all clouds.

The author, who is a member of the scientific staff of the Medical Research Council, is indebted to Dr. B. M. Wright for reading a summary of this paper at the Lisbon conference, and for much valuable criticism and discussion of its subject matter.

References

Bedford, T., and Warner, C. G. (1950). *British Journal of Industria Medicine*, **7**, 187.
Brown, J. H., Cook, K. M., Ney, F. G., and Hatch, T. (1950). *Amer. J. publ. Hlth.*, **40**, 450.
Cartwright, J., and Nagelschmidt, G. (1951). *Safety in Mines Res. Estab. Res. Rep.*, No. 24. Sheffield.
Davies, C. N. (1949). *British Journal of Industrial Medicine*, **6**, 245.
Dennis, W. L., and Sawyer, K. F. (1950). Unpublished Rep. Ministry of Supply.
Gaddum, J. H. (1941). *Ibid.*
Gessner, H., Rüttner, J. R., and Bühler, H. (1949). *Schweiz. med. Wschr.*, **79**, 1241 and 1258.
Hatch, T. F. (1950). I.L.O. 3rd Int. Conf. of Experts on Pneumoconiosis. Sydney, Australia.
Landahl, H. D. (1950). *Bull. Math. Biophysics*, **12**, 42.
——, and Herrmann, R. G. (1948). *J. industr. Hyg.*, **30**, 181.
——, Tracewell, T. N., and Lassen, W. H. (1951). *Arch. industr. Hyg.*, **3**, 359.
McCartney, J. T. (1945). U.S. Bur. Mines Rep. Invest., No. 3827.
Sharpe, J. W., and Hounam, R. F. (1950). *Proc. Conf. Electron Microscopy*, p. 186. Delft.
Walton, W. H. (1950). Private communication.
Wijk, A. M. van, and Patterson, H. S. (1940). *J. industr. Hyg.*, **22**, 31.
Wynn, A. H. A., and Dawes, J. G. (1951). *Safety in Mines Res. Establ. Res. Rep.*, No. 28. Sheffield.

Brit. J. industr. Med., 1952, **9**, 127.

THE INCIDENCE OF BLADDER TUMOURS IN A DYESTUFFS FACTORY*

BY

T. S. SCOTT

From the Clayton Aniline Company Ltd., Manchester

The incidence of bladder tumours in workers in the dyestuffs industry was first described by Rehn in 1895 and 1906. Leuenberger reported 41 cases in 1912, and by the third decade of this century in Europe exposure to beta naphthylamine and to benzidine had been accepted as a cause of bladder tumours by Kennaway (1924), Berenblum and Bonser (1937), Goldblatt (1949), and Müller (1951). In the U.S.A. Gehrmann, Foulger, and Fleming (1949) are less certain of the carcinogenicity of benzidine than the European investigators and have suggested that it cannot be accepted as a bladder carcinogen until it is known that benzidine workers without any exposure to beta naphthylamine have developed tumours of the bladder.

This paper describes 23 cases of papilloma and carcinoma of the bladder among workers exposed only to benzidine, and reviews in all 66 cases not previously reported of confirmed bladder tumours arising in a dyestuffs factory. Fifty-eight cases were found in the section where intermediates are manufactured and eight in the colour factory where the intermediates are handled. No cross transfers have taken place and the populations have remained distinct. Only the men for whom data are complete have been included in this series. In addition there are 11 former employees believed to have died of bladder tumour whose histories are not yet fully traced, and two men reported to have developed tumours with whom contact has been temporarily lost. A voluntary system of compensation, by *ex gratia* payments, should help to ensure that the histories of men who develop tumours after leaving the factory will be known.

Manufacture of Intermediates

In Table 1 it will be seen that of the 58 patients 19 have died. All except three of these 58 patients have been employed in the manufacture of benzidine, alpha naphthylamine, or beta naphthylamine, or in various combinations of the three. Twenty-three had worked solely on benzidine in a separate building and had never had contact with naphthylamines. Fifteen had worked solely on beta naphthylamine and had not had contact with benzidine. The 16 mixed exposures arose either in the still house, where benzidine and naphthylamines were sent to be distilled, or in the sulphonation building where alpha and beta naphthylamine were sulphonated. The percentage of beta isomer in the alpha naphthylamine was about 4. There have been no transfers between these departments. Three cases arose in men with other exposures.

TABLE 1

CASES OF TUMOUR OF THE BLADDER IN THE MANUFACTURE OF INTERMEDIATES

Type of Exposure	Total	Alive	Dead
Benzidine	23	15	8
β Naphthylamine ..	15	9	6
α Naphthylamine ..	1	1	0
Mixed benzidine α & β naphthylamine	16	11	5
Other	3	3	0
Total	58	39	19

Benzidine.—Benzidine manufacture by the zinc reduction method was started in 1918. It was first produced as the sulphate which was then converted to the base, and until 1944 either the sulphate, the base, or both have been isolated. Since 1944 the hydrochloride has been made. These and other changes in the process are shown diagrammatically in Fig. 1. Ortho-dichlorbenzene was used as a diluent until 1926 when it was replaced by a solvent, paraffin oil. In 1947 solvent naphtha was substituted

* A summary of this paper was read at the Tenth International Congress of Industrial Medicine, Lisbon, September, 1951.

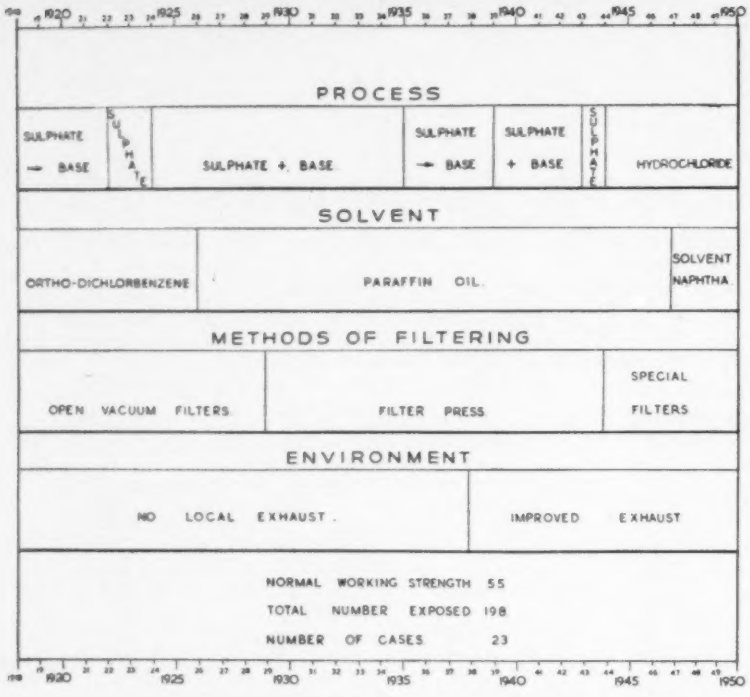


FIG. 1.—Diagram of history of benzidine manufacture from 1918 to 1950.

for the paraffin oil. Open vacuum filters were used until 1929 when they were replaced for technical reasons by a large filter press. In 1944 specially designed vacuum filters were installed. In 1938, after the first cases of bladder tumour had occurred, an exhaust ventilation system was installed. This has since been improved from time to time.

The usual number of men employed in the building housing this plant is about 55, of whom a special squad of six now work the benzidine process; the total number who have been exposed in this building for six months or more over the whole period is 198. Since 1935 there have been 23 cases none of which has been exposed to naphthylamines.

Beta Naphthylamine. — The manufacture of beta naphthylamine was started in 1920. It was manufactured in one building, distilled in another (where benzidine was also distilled for many years), and sulphonated in a third. Apart from the still house, with a normal complement of two men, the workers on these processes were never exposed to benzidine.

The process is in two stages and is shown diagrammatically in Fig. 2. The first, heating beta naphthylamine with ammonium sulphite and ammonia in autoclaves, varied little from the beginning. The second stage, that is the separation of the amine from the autoclave charge, was improved from time to time. From 1920 to 1933 the charge was filtered, stoved, distilled, set in open trays, broken up by hand, and ground. From 1933 to 1938 stoving was omitted and a flaker replaced the trays and the grinding. From 1938, when the hazard was apparent, all washing and drying was carried on in closed vessels, and molten beta naphthylamine was piped to the still, distilled and flaked. In 1947 the

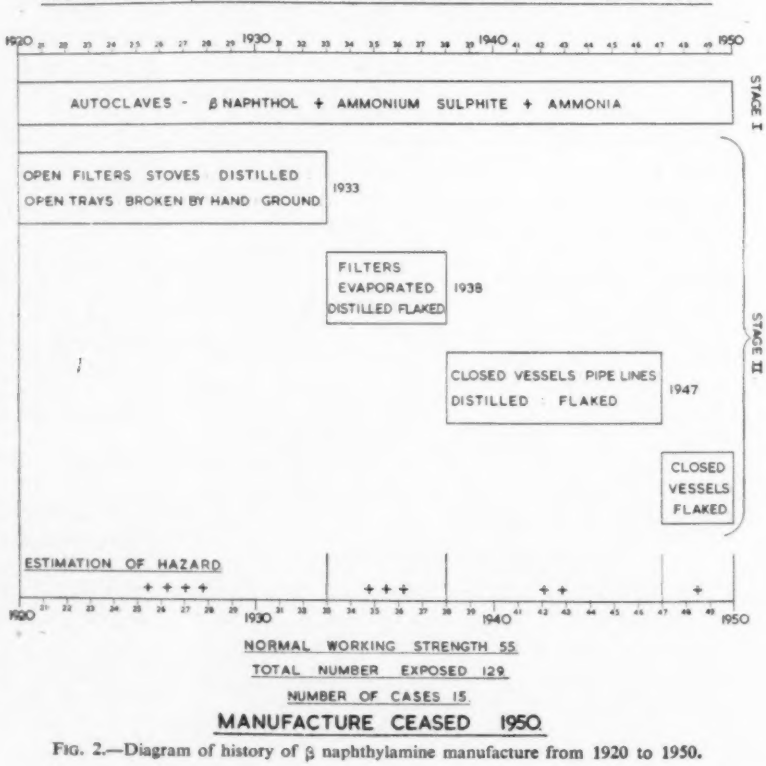


FIG. 2.—Diagram of history of β naphthylamine manufacture from 1920 to 1950.

product was flaked without distillation. After separation the amine was then sulphonated in another building.

The manufacture of beta naphthylamine and its handling in the colour factory ceased in 1950 because of the health hazard involved. The normal working complement was about 55 men, and the total number exposed for six months or more since 1920 was 129. Fifteen cases of tumour have occurred in this group since 1934.

Alpha Naphthylamine.—Alpha naphthylamine was manufactured in this factory for four years only, from 1926 to 1930, by the nitration of naphthalene and subsequent iron reduction of alpha-nitro-naphthalene. The usual number of men employed on this process was three, and the total number who worked on it for six months or more was four. None of these men has developed bladder tumour.

One case, which might be attributed to alpha naphthylamine in the manufacture of intermediates, developed a tumour after being employed for 18 years in the manufacture of naphthionate of soda from alpha naphthylamine. During this process a distillation residue containing about 17% of beta naphthylamine (from the 4% beta impurity) was recovered as a molten product, set in trays, broken up by hand, and transferred to casks. It has previously been suggested by other investigators (Goldblatt, 1949; Müller, 1951) that the beta naphthylamine impurity in alpha naphthylamine is the causative factor in tumour production, and this man was, owing to the nature of the process, more heavily exposed to the residue with a high beta naphthylamine content than to technical alpha naphthylamine.

Aniline.—The manufacture of aniline by the iron reduction process started before 1900 and has always been carried on in separate buildings. The total number exposed over six months since 1920 is 76. Previous records are not dependable. The normal working complement is 11. No cases of bladder tumour have been found among men in this process although some men have worked since the early days of primitive plant and bad conditions, and some exposures up to 45 years are substantiated. In Europe tumours attributed to aniline have been reported by Gross (1940) 33 cases, Goldblatt (1949) three cases, and Müller (1951) 22 cases, but Gehrmann and others (1949) reported that they had found no tumours in men from an aniline plant with a substantial number of employees who had worked thus exposed up to 25 years.

Other Exposures.—One man was employed on the manufacture of beta naphthylamine for three months, and for the next 27 years worked as a boiler fireman in the factory, an occupation which did not bring him into contact with intermediates. Müller (1951) reports the case of a stoker who developed bladder carcinoma after three years' exposure to firing anthracene vapours, and quotes Rosner, Young, Russell, and O'Donovan as reporting bladder carcinoma in addition to skin and scrotal carcinoma in tar and fuel operatives. The observations of Henry, Kennaway, and Kennaway (1931) do not show any increased incidence for this class for the 1921–28 period in England and Wales. As no other exposure under four years is known to have occurred in this series, he has not been included with the beta naphthylamine cases, although exposures as short as six months have been accepted by Gross (1940).

Another, a process worker, developed a papilloma after 28 years on the manufacture of ortho-toluidine and chlor-toluidines. He had no contact with benzidine. The nearest process to him in this building, however, was one in which alpha naphthylamine sulphate, which contained about 4% beta naphthylamine sulphate, was baked to make naphthionic acid. No other cases have arisen in this shed.

The third, a process worker, developed carcinoma of the bladder after working for five years on the manufacture of amino salicylic acid. In the next unit in the same building dianisidine was made. Neither of these substances has been reported previously as causing bladder tumours, although Perlman and Staehler (1933) included dianisidine in a list of substances which they mentioned as possible causes.

Exposure and Latent Periods.—The shortest period of exposure, excluding the one doubtful case

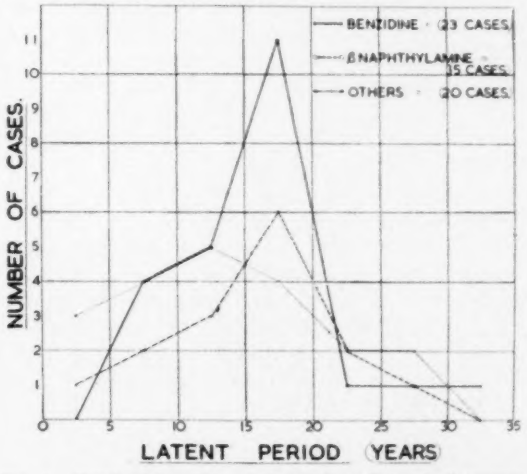


FIG. 3.—Graph of time from starting work to diagnosis of tumours.

of the boiler fireman, was four years and the longest 32 years, with an average of 16 years for all the manufacturing group. The latent period (Fig. 3), that is the time from starting work to diagnosis of tumour, showed no appreciable difference between the 23 benzidine cases with a range of eight to 32 years (average 15·9 years) and the beta napthylamine cases with a range of five to 27 years (average 15·5 years). The 20 mixed and other exposures with a range of seven to 22 years tended on the whole to appear earlier but their average latent period was 16 years (Fig. 3).

It is worth noting that, of the men exposed to beta naphthylamine who developed tumour, the one with the longest latent period (27 years' exposure) was the only chemist in the series. As a chemist he would have had considerably less exposure than a process worker.

Handling of Intermediates

All of the eight cases reported in the colour factory have arisen among men who have worked on diazotization processes in colour synthesis. Seven have handled benzidine, and one both benzidine and alpha naphthylamine with the former as the major exposure. There have been no deaths among these men (Table 3). This factory, which opened in 1919, has used benzidine in one building and small amounts of alpha naphthylamine in another. The amount of benzidine used has increased eightfold over the past 31 years and has utilized practically the whole output from the intermediates section. Special precautions were extended in this factory at about the same time that cases arose among men manufacturing intermediates, but the first case did not arise in the colour factory until 1941. Since then only one man at a time has been handling and charging benzidine. The three men so employed consecutively have been volunteers. The first, who had 14 years' previous exposure to benzi-

TABLE 2

TYPES OF TUMOUR ENCOUNTERED IN THE MANUFACTURE OF INTERMEDIATES

Type of Exposure	Total Cases	Papilloma	Carcinoma	Papilloma Carcinoma
Benzidine	23	14^{4}	7^{4}	2
β Naphthylamine	15	8^{1}	7^{5}	—
α Naphthylamine	1	—	1	—
Mixed benzidine and α & β naphthylamine ..	16	5^{2}	9^{3}	2
Other	3	2	1	—
Total	58	29^{7}	25^{12}	4

The index figures relate to deaths.

Eight men left the firm and subsequently developed tumours. The period of exposure was from four to 11 years with an average of seven years. The average latent period ranging from nine to 20 years was 15 years, which is similar to the latent period for those who had not left the firm before developing tumours.

Types of Tumour.—Of the 58 cases, 29 had papilloma, 25 had carcinoma, and four had papilloma which recurred as carcinoma (Table 2). There is no striking difference in the incidence of benign and malignant tumours in the varying types of exposure in these small numbers, except that the men with mixed exposures had a higher proportion of carcinomata than other groups.*

* The pathology and treatment of these tumours will be the subject of another paper n collaboration with Mr. Poole-Wilson.

TABLE 3

CASES OF TUMOUR OF THE BLADDER IN HANDLING OF INTERMEDIATES (COLOUR FACTORY)

Type of Exposure	Total	Alive	Dead
Benzidine	7	7	0
Benzidine + α naphthylamine	1	1	0
Total	8	8	0

Exposure period ranged from 15 to 27 years (average 21 years)

Normal working strength .. 14
Total numbers exposed .. 86
No. cases 8

dine, developed a tumour a year later and was taken off at his own request ; the second was promoted to foreman after six years ; and the present one has handled and charged all the benzidine for three years. The latter two men have not developed tumours.

The normal working complement on the benzidine colours is 14 and the total number who have worked six months or more since 1919 is 86.

Exposure and Latent Periods.—All the men in this factory who developed tumours were still employed at the time of diagnosis and therefore the latent and exposure periods are the same. The shortest was 15 years and the longest 27 years with an average of 21 years. The cases among the men handling benzidine in the colour factory had a longer average exposure (21 years) than those manufacturing (16 years), and the first case among the handlers arose seven years later. It is reasonable to assume, therefore, that the intensity or the nature of the exposure in the manufacturing process was a determining factor in producing tumours earlier than in handling.

Age at Diagnosis of Bladder Tumours

Of the 66 cases, 40 entered the industry before the age of 30, 17 between the ages of 30 and 40, and nine after the age of 40. It will be seen from Table 4 that of those under 30 the average age of onset of tumour was 42 and of death was 44 ; of those between 30 and 40 the average age of onset was 51 and of death 54 ; and in those over 40 the average age of onset was 58 and of death 66. From the Registrar General's figures for 1947 the average age of death of 1,417 males in England and Wales who died of neoplasms of the bladder, urethra, and ureter was 67·5 years. Stocks (1950) took records of cases of cancer of the bladder and ureter from 275 hospitals in England and Wales in 1945. He found that the average age of all cases at first attendance was 62·3 years and at death 66·3 years. These figures emphasize the shortened expectation of life of men who are exposed to carcinogens before the age of 40 and develop bladder tumour. Thus, there is a strong argument in favour of excluding young entrants from processes where the hazard of bladder carcinogens can even be suspected. It has been the policy in recent years in this factory to debar men under 30 from such plants and to prefer healthy men over 40.

TABLE 4

AGE AT DIAGNOSIS OF BLADDER TUMOURS (ALL CASES)

Age on Entry	Under 30	30–40	Over 40
Number of cases	40	17	9
(Number of deaths)	(10)	(5)	(4)
Average age at diagnosis of tumour	42	51	58
(Range)	(25–52)	(41–62)	(47–66)
Average at death	44	54	66
(Range)	(34–51)	(51–60)	(61–69)

Future Prevention

Methods of prevention at first followed the usual lines. Technical improvements in the plants and processes, some of which have been indicated, have been applied. Men are carefully selected by pre-employment examination with consideration given to age, family history, and previous health ; strict hygienic measures such as special working clothing and daily bathing are obligatory ; regular medical supervision is carried out ; and although routine periodic cystoscopy of all workers is not yet applied, routine examination of the urine for red and white blood cells is done on all workers monthly. But with the possibility of such a high morbidity and early mortality arising from these processes more radical measures were deemed to be essential. Delayed by the war with its greater priorities, a new benzidine plant in which all possible precautions are taken to obviate contact between the operator and the carcinogens has now been built on a new site and will be in production this year. The old plant will be pulled down. Apart from a small number of key men, whose experience is needed and who will be selected from those who have had tumours and are cured, new men from the older age groups, with no previous exposure, will be selected and trained for this job. Thus, should any of these new men develop tumours it will be possible to attribute them solely to exposure on this plant when assessing the incidence.

Beta naphthylamine manufacture ceased a year and a half ago, as soon after the war as technical exigencies permitted, and sulphonation of beta naphthol followed by amidation is now the method used for preparing beta naphthylamine sulphonic acids in the synthesis of the colours for which beta naphthylamine was previously used as an intermediate.

In the future it should be possible to know whether or not these hazards have been controlled, but the time necessary for the development of tumours may be so long that we cannot be sure that the problem has been mastered until a whole generation has been unaffected. A list has been compiled, and is kept up to date, of all men who are known to have worked more than six months with carcinogens

since 1918. As even shorter exposures may come to be accepted as sufficient to initiate a tumour, careful records are now kept of any exposure, however short, and no cross transfers are permitted between departments where carcinogens are manufactured or used.

Summary

Sixty-six cases of bladder tumours in a dyestuffs factory are reported.

The methods and history of manufacture and handling are described.

Of the 66 cases there were 30 (23 in the manufacturing section and seven in the handling section) whose exposure was to benzidine, and which had never been exposed to beta naphthylamine. Benzidine is therefore considered to be as dangerous a carcinogen, industrially, as beta naphthylamine.

The role of beta naphthylamine as a bladder carcinogen in manufacture and experimentally has previously been accepted, and 15 cases are attributed to it alone and others to mixed exposures. Beta naphthylamine as an impurity cannot be eliminated as a cause of bladder tumours in men who worked on alpha naphthylamine.

No cases have been traced which can be attributed to aniline, despite the longer time of manufacture and periods of exposure, and the similar numbers engaged on it compared with benzidine and beta naphthylamine.

Three other exposures are described in which the cause of tumour was doubtful or unknown.

Exposure, latent periods, and ages at diagnosis and death are discussed.

Men under the age of 30 should not be employed in the manufacture or handling of substances known or suspected to be bladder carcinogens and, when possible, healthy men over 40 should be employed on these processes.

This paper would not be complete without an expression of gratitude to Mr. J. B. Macalpine and his successor Mr. D. S. Poole-Wilson, the urologists who have had these cases under their care.

REFERENCES

Berenblum, I., and Bonser, G. M. (1937). *J. industr. Hyg.*, **19**, 86.
Gehrmann, G. H., Foulger, J. H., and Fleming, A. J. (1949). Proc. 9th int. Congr. Industr. Med. London, 1948, p. 472. Bristol.
Goldblatt, M. W. (1949). *British Journal of Industrial Medicine*, **6**, 65.
Gross, E. (1940). *Angew. Chem.*, **53**, 368.
Henry, S. A., Kennaway, N. M., and Kennaway, E. L. (1931). *J. Hyg., Camb.*, **31**, 125.
Kennaway, E. L. (1924). *Brit. med. J.*, **1**, 564.
Leuenberger, S. G. (1912). *Bruns'. Beitr. klin. Chir.*, **80**, 208.
Müller, A. (1951). *Helv. chir. Acta*, **18**, 1.
Perlmann, S., and Staehler, W. (1933). *Z. urol. Chir.*, **36**, 139.
Rehn, L. (1895). *Arch. klin. Chir.*, **50**, 588.
——, (1906). *Verh. dtsch. Ges. Chir.*, **35**, 313.
Stocks, P. (1950). "Cancer Registration in England and Wales," *Studies in Medical and Population Subjects*, no. 3. H.M.S.O. London.

Brit. J. industr. Med., 1952, **9**, 133.

URINARY COPROPORPHYRINS IN LEAD POISONING

BY

J. E. KENCH, R. E. LANE, and H. VARLEY

From the Nuffield Department of Occupational Health, University of Manchester, and the Department of Clinical Pathology, Manchester Royal Infirmary

(RECEIVED FOR PUBLICATION DECEMBER 27, 1951)

Many aspects of haemopoiesis in lead poisoning remain obscure. When industrial plumbism was common the haemolytic action of lead was frequently described (Aub, Fairhall, Minot, and Reznikoff, 1925), although there were atypical features. The erythrocytes were somewhat deficient in haemoglobin, and faecal urobilinogen was not increased. Lead poisoning also differs from true haemolytic anaemia in the occurrence of coproporphyrin III in the urine (Grotepass, 1932; Fischer and Duesberg, 1932; Watson, 1936). It is coproporphyrin I which is excreted in haemolytic conditions characterized by hyperplasia of the bone marrow (Watson, 1937; Dobriner and Rhoads, 1938; Grinstein, Kamen, Wikoff, and Moore, 1950).

Rimington (1936) advanced the view that in enzymic synthesis of the porphyrins, in addition to type III porphyrin used in haemoglobin formation, a small amount of type I series is always produced, which, being of no value, is excreted. Urinary coproporphyrin I excretion therefore provided an index of the rate of haemopoiesis. Rimington (1938) further postulated that the coproporphyrin III excretion in lead poisoning is due to a partial blockage by lead of the entrance of iron into protoporphyrin 9, thus interfering with haemoglobin formation (Fig. 1). He discounted the haemolytic action of lead, since faecal urobilinogen was normal, but recent isotopic studies by London, Shemin, and Rittenberg (1948) on the conversion *in vivo* of glycine N^{15} to stercobilinogen have shown that the latter is a very unreliable index of the rate of degradation of circulating haemoglobin. Kench, Gillam, and Lane (1942) found that excretion of coproporphyrin III was not sufficient by itself to account for the degree of haemoglobin deficit in this disease, and suggested that lead interfered all along the line of enzymatic syntheses, causing restricted formation of porphyrin and other intermediates.

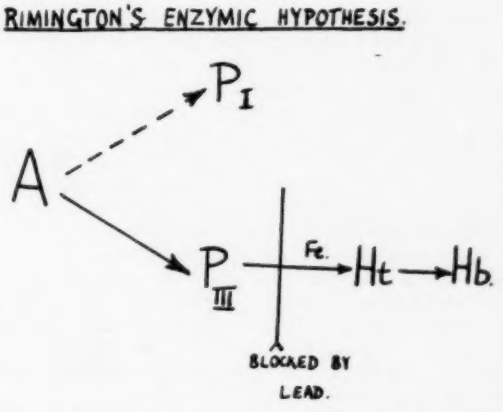


FIG. 1.—Rimington's enzymic hypothesis: A = pyrrole precursors. P_I and P_{III} = porphyrin series I and III. Ht = haem.

McFadzean and Davis (1949), by studying the effect of splenectomy in lead-poisoned rabbits, concluded that lead has a toxic action upon erythrocyte precursors in the bone marrow, producing defective haemoglobinization and persistence of basophilic substance in granular form, associated with an increased iron content. The stippled erythrocytes which arise from these precursors on entering the circulation are rapidly taken up by the spleen, producing a haemolytic type of anaemia, the severity of which varies with the intensity and duration of lead absorption.

In this paper we describe a study of the excretion of lead and porphyrins in the urine of seven bath enamellers immediately after considerable exposure to lead for three to six months. They were first seen in December, 1950, and had signs and symptoms of poisoning including anaemia, constipation, and in two cases severe abdominal colic. They were withdrawn from lead work immediately, but subsequently four of them did lead smelting without the knowledge of the works manager.

We have observed that coproporphyrin III excretion in lead poisoning is accompanied by an increase in coproporphyrin I, which supports the conclusion of McFadzean and Davis that there is a haemolytic action in lead poisoning.

Materials and Methods

Haemoglobin was determined by the M.R.C. grey wedge photometer (MacFarlane, King, Wootton, and Gilchrist, 1948).

Punctate basophilia was counted under dark ground illumination (Lane, 1949).

Urinary lead was determined by a modified dithizone method (Kench, 1940) on specimens collected twice weekly.

Ether-soluble porphyrins were extracted by the usual acetic acid-ether procedure and measured fluorimetrically (Rimington, 1943). The urinary residue was shaken with alumina to adsorb uroporphyrins, with subsequent elution as methyl esters. Ether-soluble porphyrins extracted from the urine of all cases were pooled. The total quantity of urine was 46 litres collected over 130 days. These porphyrins were analysed by single dimensional chromatography by the method of Nicholas and Rimington (1949).

Chromatography of the derived methyl esters was performed as described by Chu, Green, and Chu (1951), with certain modifications. The solvents were purified as follows: oleic acid was distilled under 15 mm. Hg pressure; n-propanol was fractionally distilled, b.p. 95–97° C.; kerosene was filtered and distilled; main fraction b.p. 140–160° C.

Whatman No. 1 filter paper was used without pretreatment. Single strip chromatograms were developed with oleic acid-kerosene (1 : 1), or with the mixtures chloroform-kerosene (2·6 : 4) and n-propanol-kerosene (1 : 5). Chloroform-kerosene, followed by n-propanol-kerosene, was also used for two-dimensional separation.

The fluorescent areas on the paper were cut out with a glass microscope slide, and the porphyrin methyl esters extracted with 4 ml. chloroform (or 4 ml. $CHCl_3$ + 1 ml. glacial acetic acid from two-dimensional chromatograms). Coproporphyrin III was diluted with chloroform until its fluorescence matched that of the coproporphyrin I solution. The positions of all spots were checked with authentic specimens of coproporphyrin I and III methyl esters, whose recovery from the chromatogram was complete, within the error of fluorescence measurement. Losses sustained by the coproporphyrin isomers in the methylation procedure were similar. Oleic acid-kerosene remaining on the paper, after allowing to drain for one minute, did not interfere with the measurement of porphyrin fluorescence, which was not quenched, and blue fluorescence was screened by the orange filter incorporated in the fluorimeter.

The urinary specimens, which were collected on the 329th and 330th days, were each examined by paper chromatography for separation of isomers.

Coproporphyrin methyl esters prepared from pooled ether-soluble porphyrins were separated by repeated rigorous extraction with cold, dry ether. The later ether washings carried away small quantities of coproporphyrin I, which were sacrificed to ensure that no trace of isomer III could remain. The crude insoluble material (7 mg.) was crystallized from methanol-chloroform to give coproporphyrin I methyl ester m.p. 244° C. (Fig. 2), whilst coproporphyrin III ester had m.p. 136° C.

Results

Anaemia present at the outset of illness had been mainly resolved by the 120th day. There was an average gain of 3·7 g. haemoglobin per 100 ml. blood (Table 1).

At first punctate basophil counts ranged from 2,000, to 13,000 per million R.B.C.s and gradually decreased,

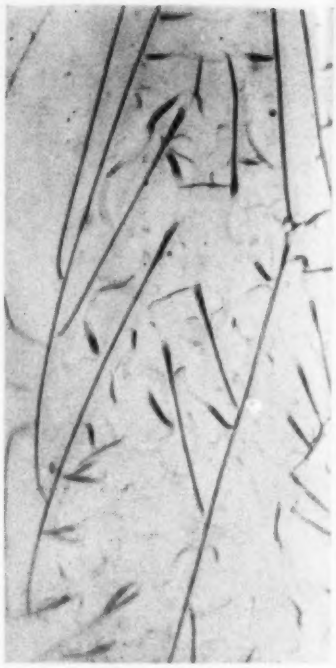


FIG. 2.—Photomicrograph of crystalline coproporphyrin I tetra-methyl ester isolated from urine in lead poisoning × 350..

TABLE 1

HAEMOGLOBIN CONCENTRATION AND PUNCTATE BASOPHILIA IN LEAD POISONING

Case No.	Day of Experiment	Haemoglobin Concentration (g./100 ml. blood)	Punctate Basophils (per million R.B.C.)
1	5	9·5	12,000 S.M.
	110	14·4	1,300 S.
	127	13·2	1,000 S.
2	26	9·2	10,000 S.M.L.
	70	15·5	7,200 S.M.L.
	127	14·9	2,200 S.
3	5	12·4	2,000 S.M.
	70	14·3	
	120	14·9	2,100 S.
4	2	12·8	4,000 S.M.
	120	14·2	2,400 S.
	127	14·3	1,600 S.
5	2	12·4	13,000 S.M.L.
	110	16·2	4,000 S.M.
	127	15·5	3,200 S.M.L.
6	6	11·9	3,500 S.M.
	60	11·0	
7	1		10,000 S.M.L.
	26	9·2	7,200 S.M.L.
	70	15·5	

S.M.L.—small, medium, large granules respectively.

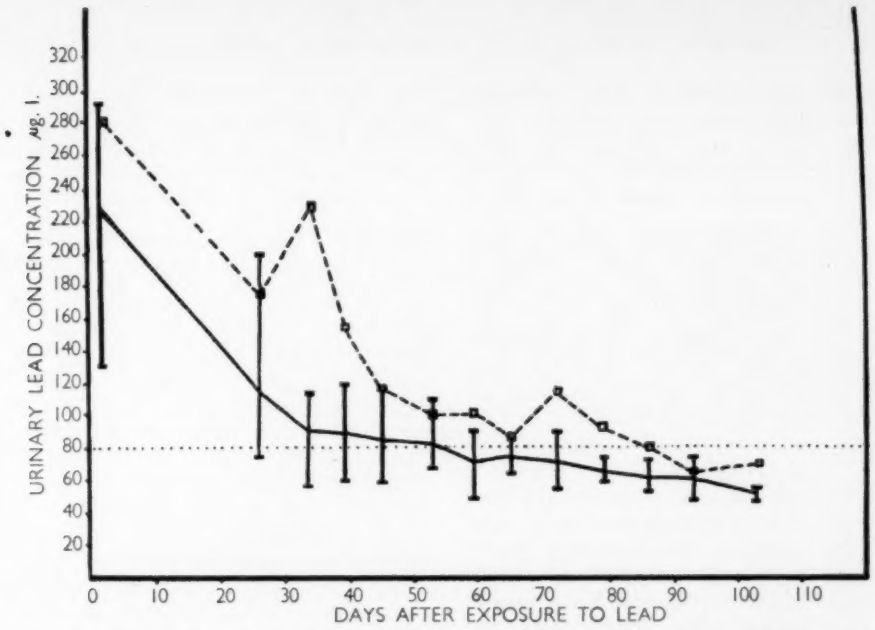


FIG. 3.—Graph illustrating changes in urinary lead in lead poisoning. The upper curve presents data for one workman with a history of protracted, heavy lead absorption (Case No. 4). The lower curve shows the mean values for the remaining six men. Vertical lines give the ranges of observations. The upper limit of normal is shown by the horizontal dotted line.

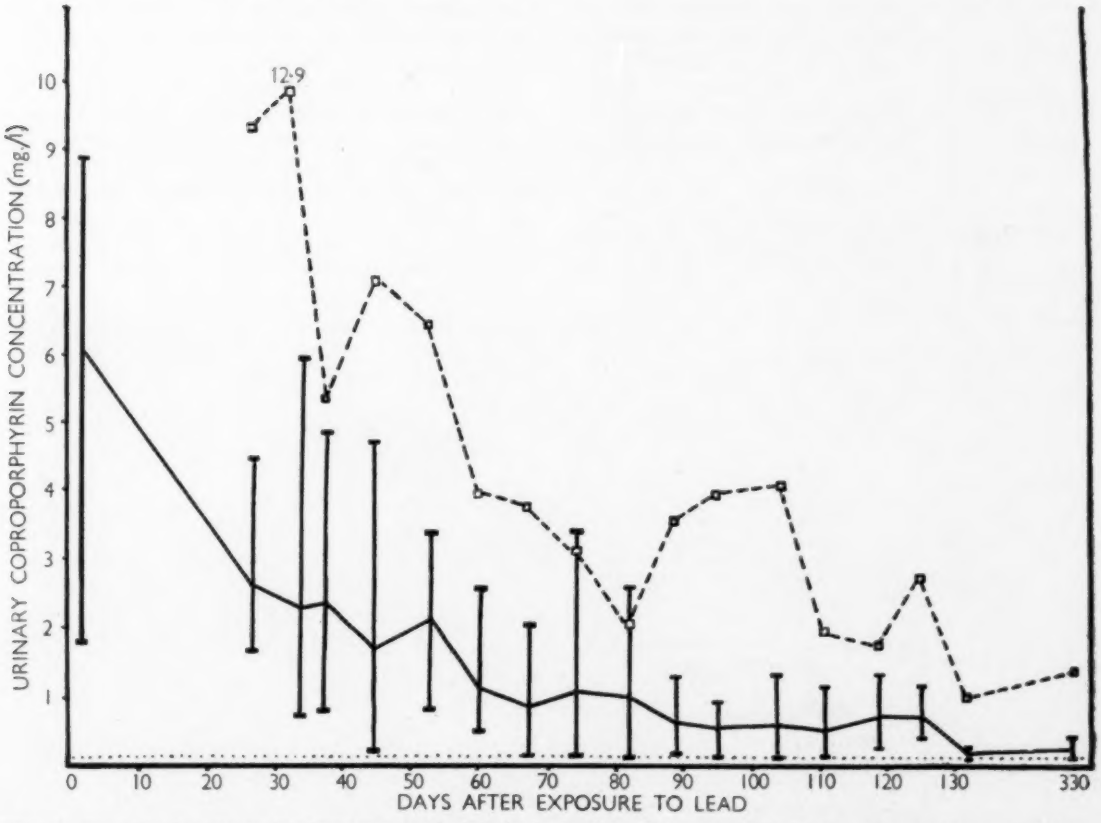


FIG. 4.—Graph showing changes in urinary coproporphyrin in lead poisoning. The upper curve is for the workman with protracted, heavy lead absorption (Case No. 4). The lower curve shows the means for the remaining six men. Vertical lines give the ranges of observations. The upper limit of normal is shown by the horizontal dotted line.

except in Case 3 whose low initial count remained unchanged. All counts after the 120th day were within the normal range of 1 to 3,000 (Lane, 1949).

Concurrently with improvement in the clinical condition and blood picture, the mean values of urinary lead and coproporphyrin concentrations reached normal limits at 55 and 133 days respectively (Figs. 3 and 4). One worker (Case 4), however, with a previous history of protracted, heavy lead absorption was excreting 1·4 mg. coproporphyrin per litre of urine 330 days after exposure. A temporary rise in porphyrin excretion at 110 days in four workmen was subsequently traced to their having done lead smelting without the knowledge of the works manager.

Single-dimensional chromatography (Nicholas and Rimington, 1949) of the pooled ether-soluble porphyrins collected during the first 130 days showed the presence of coproporphyrin only. Examination of the derived methyl esters by chromatography using oleic acid-kerosene 1 : 1 (Chu, Green, and Chu, 1951) showed that these comprised a mixture of coproporphyrin I (30%) and coproporphyrin III (70%).

FIG. 5.—Chromatogram of porphyrin methyl esters prepared from urine in lead poisoning. Photograph of spots red-fluorescent in ultra-violet light. Solvent oleic acid-kerosene (1 : 1).

TABLE 2

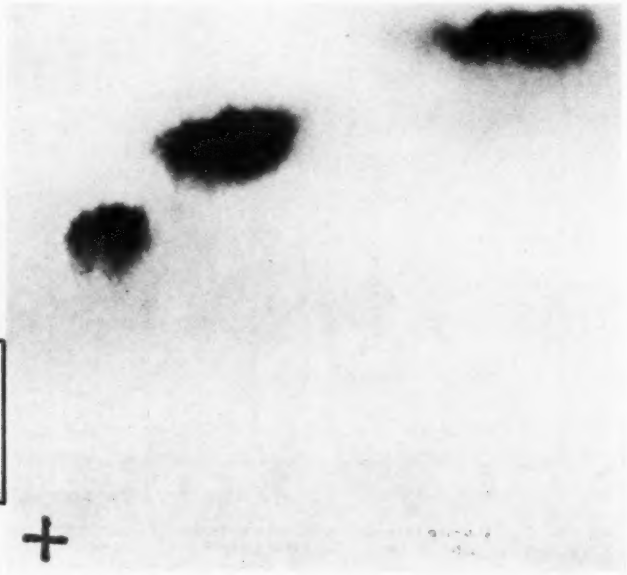
URINARY COPROPORPHYRINS I AND III IN LEAD POISONING 330 DAYS AFTER EXPOSURE

Case No.	Coproporphyrin I		
	Rf.	Proportion in Mixture (%)	Urinary Concentration (μg./l.)
1	0·42	47	67
2	0·44	40	42
3	0·42	29	117
4*	0·41	20	348
	Coproporphyrin III		
1	0·64	53	76
2	0·67	60	62
3	0·69	71	285
4*	0·67	60	1045

* This specimen also contained an unidentified porphyrin Rf.0·84, 20% of total porphyrin concentration. Data in this table are means of observations on urinary specimens collected on the 329th and 330th days.

Urinary specimens collected on the 329th and 330th days were examined (Figs. 5 and 6). Coproporphyrin I was present in each specimen, and ranged from 20–47% of the total porphyrin concentration (Table 2). It represented 28% of the pooled material.

Uroporphyrin was not detected in any of the ether-extracted residues.



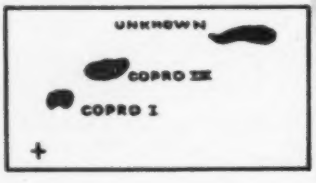


FIG. 6.—Two-dimensional chromatogram of porphyrin methyl esters prepared from urine in lead poisoning. Photograph of spots red-fluorescent in ultra-violet light. Solvents chloroform-kerosene (2·6 : 4) and n-propanol-kerosene (1 : 5).

For comparison eight specimens of normal urine contained 38, 37, 28, 90, 71, 17, 17, and 52 μg. per litre with a mean value of 44 μg. coproporphyrin per litre. On the Chu chromatogram coproporphyrin I constituted 55% of the total porphyrin.

Discussion

Forty-six litres of urine were collected during tne first stage of the study, and contained 86·7 mg. ether-soluble porphyrins of which 30% was coproporphyrin I. If the coproporphyrin I had been normal it would have contributed only 1·6% of the total urinary porphyrin. As this result was entirely unexpected, further specimens were collected on the 329th and 330th days and these confirmed that an increase in total urinary porphyrin was accompanied by a rise in coproporphyrin I. We have calculated that when the bath enamellers had acute lead poisoning they were excreting approximately 1 mg. of coproporphyrin I per litre of urine, which, with the high punctate basophil count, is consistent with bone marrow hyperplasia. This hyperplasia would arise in response to an increased rate of breakdown of abnormal red blood cells, and, indeed, the workman with the heaviest lead absorption (No. 4) had a serum bilirubin level of 2 mg. per 100 ml. (indirect). However, it is evident that lead also interferes with haemoglobin production within the erythrocyte precursors, probably inhibiting many enzymes taking part in the synthesis of intermediates, including the formation of haem by incorporating iron into protoporphyrin 9. This disturbance of porphyrin metabolism leads to loss of coproporphyrin III in the urine.

Lead has, therefore, two toxic actions in haemopoiesis, which together are responsible for the anaemia. (1) It interferes with normal maturation of erythroid marrow cells leading to the production of so-called stippled cells which are rapidly broken down by the reticulo-endothelial system. (2) Haemoglobin synthesis in the precursor cells is inhibited, probably by a general action of lead on the enzymic formation of porphyrin and other intermediates.

Coproporphyrin I excretion appears to be associated with hyperplasia of the erythroid marrow cells in haemolytic jaundice, in congenital porphyria (Aldrich, Hawkinson, Grinstein, and Watson, 1951), and in lead poisoning, and is apparently independent of haemoglobin formation. It seems probable that the coproporphyrin isomers I and III arise by independent syntheses from their common precursor glycine. The possibility that coproporphyrin I may play a vital role in cell proliferation awaits further study.

This hypothesis is presented in an attempt to resolve the apparent anomalies of coproporphyrin I and III formation in lead poisoning, but it does not exclude the long recognized action of lead on the erythrocytes.

The practical issues involved in the use of urinary porphyrin excretion as an index of lead absorption will be discussed in a further communication.

Summary

A study has been made of urinary lead and coproporphyrin in seven cases of lead poisoning.

Accompanying the well recognized increased urinary excretion of coproporphyrin III in lead poisoning there was a concomitant increase of coproporphyrin I at a mean of 30% of the total porphyrin excretion.

It is suggested that coproporphyrins I and III are synthesized independently. Coproporphyrin I is associated with proliferation of marrow cells as distinct from coproporphyrin III, which is linked with the formation of haemoglobin within the cells.

We should like to thank Mr. S. Coxon and Mr. C. Howe for their valuable assistance in this work.

References

Aldrich, R. A., Hawkinson, V., Grinstein, M., and Watson, C. J. (1951). *Blood*, **6**, 685.
Aub, J. C., Fairhall, L. T., Minot, A. S., and Reznikoff, P. (1925). *Medicine, Baltimore*, **4**, 1.
Chu, T. C., Green, A. A., and Chu, E. J. (1951). *J. biol. Chem.*, **190**, 643.
Dobriner, K., and Rhoads, C. P. (1938). *J. clin. Invest.*, **17**, 105.
Fischer, H., and Duesberg, R. (1932). *Arch. exp. Path. Pharmak.*, **166**, 95.
Grinstein, M., Kamen, M. D., Wikoff, H. M., and Moore, C. V. (1950). *J. biol. Chem.*, **182**, 715.
Grotepass, W. (1932). *Hoppe-Seyl. Z. physiol. Chem.*, **205**, 193.
Kench, J. E. (1940). *Biochem. J.*, **34**, 1245.
——, Gillam, A. E., and Lane, R. E. (1942). *Ibid.*, **36**, 384.
Lane, R. E. (1949). *British Journal of Industrial Medicine*, **6**, 125.
London, I. M., Shemin, D., and Rittenberg, D. (1948). *J. clin. Invest.*, **27**, 547.
Macfarlane, R. G., King, E. J., Wootton, I. D. P., and Gilchrist, M. (1948). *Lancet*, **1**, 282.
McFadzean, A. J. S., and Davis, L. J. (1949). *Quart. J. Med.*, **18**, 57.
Nicholas, R. E. H., and Rimington, C. (1949). *Scand. J. clin. Lab. Invest.*, **1**, 12.
Rimington, C. (1936). *Onderstepoort J. vet. Sci.*, **7**, 567.
——(1938). *C. R. Lab. Carlsberg. Sér. Chim.*, **22**, 454.
——(1943). *Biochem. J.*, **37**, 137.
Watson, C. J. (1936). *J. clin. Invest.*, **15**, 327.
——(1937). *Ibid.*, **16**, 383.

Brit. J. industr. Med., 1952, **9**, 138.

STUDIES ON COTTON DUST IN RELATION TO BYSSINOSIS

PART I: BACTERIA AND FUNGI IN COTTON DUST

BY

G. FURNESS* and H. B. MAITLAND

From the Department of Bacteriology, University of Manchester

(RECEIVED FOR PUBLICATION JANUARY 28, 1952)

There is general agreement that byssinosis is associated with the inhalation of cotton dust. Good descriptions of the disease have been given by the Home Office Departmental Committee on Dust in Card Rooms in their report (1932), by Prausnitz (1936), and by Schilling (1950). An extensive and complete review of the literature on cotton dust in relation to affections of the respiratory tract has been prepared by Caminita, Baum, Neal, and Schneiter (1947). The dust affects the lungs, and the first symptoms are cough, a feeling of tightness in the chest, and breathlessness which develop gradually after working for several years in the dusty atmosphere produced by opening bales of cotton and by the carding machines which clean and comb-out the fibres. Some persons do not suffer from this disease after a life-time of exposure to the dust; those who do, find their symptoms are worse on Mondays after a week-end away from the mill. The disease is usually progressive, but some workers suffer only on Mondays during the whole of their working lives, without any progression. A change of occupation which avoids exposure to the dust gives freedom from symptoms, but a return to the dusty atmosphere of the mill after such a period of freedom results immediately in a return of symptoms. The pathological changes found in the later stages are those of emphysema. Where the disease progresses symptoms eventually persist throughout the whole week and finally there is incapacity for work, frequently with congestive heart failure.

The respiratory disability has been termed "asthma", and the disease has been known as "card-room asthma", "strippers and grinders asthma", or "cotton mill asthma", a terminology which has been used in a loosely descriptive sense but which nevertheless has had some influence in suggesting, by analogy with some other types of asthma, that a factor of sensitization may be implicated in the aetiology of the disease, an idea that has been strengthened by the occurrence of symptoms on exposure to the dust on Mondays or after a spell away from the mill. Other features can be made to fit this hypothesis: the period of exposure which elapses before symptoms begin might indicate the time required for sensitization to develop; difference in individual susceptibility to byssinosis might be due to inherent differences in the proneness to become sensitive; the fact that in the early stages of the illness symptoms are worse on Mondays and decrease or disappear later in the week might mean a temporary partial desensitization. There is, however, nothing conclusive or convincing in such reasoning and other explanations for the peculiarities of byssinosis are possible.

The fraction of the dust concerned in producing the disease and the mechanism of its action are still not clearly understood. Prausnitz (1936) stated that byssinotics were hypersensitive, as judged by skin tests, to a substance which could be extracted from cotton dust and concluded that allergy was a factor in aetiology. The dust was also said to contain an unidentified irritant or toxic fraction which may be different from the fraction that was considered to cause hypersensitiveness. The effect of the toxic fraction on the lungs was believed to be of primary importance. In addition histamine or a similar substance has been identified in extracts of cotton dust (Maitland, Heap, and Macdonald, 1932; Macdonald and Maitland, 1934; Macdonald and Prausnitz, 1936; Haworth and Macdonald, 1937). The histamine has been shown to come from the fragments of cotton-seed, both kernels and husks, in the dust (Macdonald and Prausnitz, 1936).

* Working under a grant from the British Cotton Industry Research Association.

As a preliminary, the bacterial and fungal content of cotton dust has been studied. Other studies on cotton dust in relation to byssinosis, particularly the possibility of specific sensitization, will be reported in other papers.

It has long been known that large numbers of bacteria and fungi are present in cotton dust but there has been little systematic investigation of this subject, apart from the work of Prindle (1934a, 1934b) who made a survey of the organisms found in freshly collected and stored commercial raw cotton in America. The flora changed on storage; in freshly collected cotton it consisted largely of non-sporing soil bacteria and fungi of the genera *Homodendrum*, *Alternaria*, and *Fusarium*, together with smaller numbers of *Penicillium* and *Aspergillus*; after storage spore-forming bacteria and the fungi *Penicillium* and *Aspergillus* predominated.

There is no evidence that infection with the bacteria or fungi found in cotton dust is a factor in the aetiology of byssinosis. The acute illness investigated by Neal, Schneiter, and Caminita (1942), Schneiter, Neal, and Caminita (1942), and Caminita, Schneiter, Kolb, and Neal (1943) which occurred among workers exposed to dust from low grade and stained cotton was a distinct entity, not connected with byssinosis. This cotton contained large numbers of *Aerobacter cloacae*, almost to the exclusion of other bacteria. The inhalation of this organism was considered to be the cause of the illness, due to the action of its endotoxin rather than to infection or sensitization. Its abundance in this cotton was associated with particular climatic circumstances and undue exposure of the cotton to contamination from the soil. Relatively few fungi were isolated from it but they comprised the genera *Alternaria*, *Mucor*, *Rhizopus*, *Fusarium*, *Sporotrichum*, *Aspergillus*, *Cladosporium*, and *Penicillium*.

Although scattered observations have furnished some general data about the kind and numbers of bacteria and fungi in dust from cotton or from the air in cotton mills there is very little information about the possibility that biologically active substances such as toxins or allergens might have their origin in these microorganisms. Examination of some fungi showed that they were not a source of histamine (Macdonald and Prausnitz, 1936).

The scope of this paper is to examine qualitatively and quantitatively the flora of a fine fraction of dust obtained from various grades of cotton received in this country.

Source and Nature of Materials Examined

Samples of dust have been separated from " cotton fly " removed from the raw cotton by the cotton-cleaning machinery and from material collected by the air-cleaning plant from the atmosphere in the vicinity of these machines. The varieties of cotton being processed included fine grade cottons of the Sudan type, medium grade cottons of the American type, and low grade cottons of the Indian type. The crude material was a mixture of dust and cotton fly in varying proportions depending on its origin; from the air-cleaning plant it consisted mainly of short fibres mixed with very fine dust; some flue dust from the scutcher had only a small quantity of fly; material from the Shirley cage * was made up of a large bulk of fibre with pockets of adhering and intermingled dust. These differences in the crude material were reflected in the character of the dust obtained from them, particularly in the relation of weight to volume. The crude material was teazed out on a 90-mesh sieve which was passed over the upturned nozzle of a " Hoover dustette " fastened to a stand. The dust was immediately recovered from the bag and stored in glass-stoppered jars. This fine fraction of dust which passed the 90-mesh sieve is referred to throughout these investigations as " cotton dust ".

The volume of dust per unit weight was estimated approximately by noting the volume of about 20 g. in a 100-ml. cylinder.

The physical character of the dust is shown in Table 1.

The moisture content of five samples was very similar, ranging from 4·0 to 6·3% and averaging less than 5%. At this low humidity any increase in the number of bacteria and moulds during storage would be unlikely to occur.

The grade of cotton being processed did not have any constant relationship to the weight per unit volume of the dust obtained from it. Dust from the air-cleaning plants was always lighter than that obtained from the cotton-cleaning machinery and its ash content was also less than that of dust from the other machines.

Microscopic Examination of Cotton Dust

Smears of watery suspensions of cotton dust were fixed by heat, defatted for 15 minutes in a bath of petroleum ether, dried, and stained or mounted appropriately. Gram-positive cocci and bacilli, and bacterial spores were present in all samples. No bacteria have been found in the lumen of fragments of fibre. Mould hyphae and

* The Shirley cage is a perforated cylinder rotating at high speed so that the cotton sucked on to its surface forms a much thinner layer than on a conventional, slow-speed cage. This permits free extraction of dust into the interior of the cage, while the cleaned cotton from its surface is enabled to go forward in a current of clean air to the next machine.

TABLE 1

SUMMARY OF SAMPLES OF COTTON WASTE EXAMINED AND PHYSICAL CHARACTERS OF THE DUST EXTRACTED

Sample*	Machine Extracting Waste	Mix of Cotton from which Waste was Extracted	Cotton Dust (g. per 100 ml.)	Moisture (%)	Ash (%)
A	Shirley cage	Sudan	45·43	4·0	70·0
B	,, ,,	½ Egyptian (Karnak) and ½ Sudan	19·7	—	—
C	Air-cleaning plant	⅓ Menoufin, ⅓ Karnak ⅓ Sudan Sakel	8·36	—	—
D	,, ,,	½ Sudan Sakel, ½ Karnak	16·36	—	—
E	Flue dust (opening and cleaning machinery)	Sudan Sakel	31·0	5·4	62·0
F	Shirley cage	⅝ American, ⅛ Brazilian, ⅛ East African	27·42	4·7	55·3
G	,, ,,	½ American, $\frac{3}{10}$ Uganda, ⅕ Sao Paulo	32·9	4·2	63·1
H	Air-cleaning plant	American	11·17	6·3	33·5
K	,, ,,	½ American, ½ Peruvian	9·93	—	—
L	Filter bags (opening and cleaning machinery)	⅜ Indian, $\frac{1}{12}$ Argentine, ¼ Turkish	35·76	—	—
M	Filter bags (opening and cleaning machinery)	½ American, ⅛ Argentine, ⅛ Turkish, ⅛ Sudan	20·43	—	—
N	Filter bags (opening and cleaning machinery)	Changing mixture of low grade American	34·0	—	—
P	Filter bags (card-room)	Pakistan Sind, Indian Dhallera and Oomras and some Turkish	37·75	—	—

* Samples A—E were from fine grade cotton, F—K from medium grade, and L—P from low grade.

mould spores were also found; the spores were similar to those of *Aspergillus niger* and a species of *Penicillium* isolated from the dust. Large quantities of fibre and debris were seen in all samples. It was not feasible to estimate the total number of bacteria, fungi, or their spores, and it was therefore not possible to determine the ratio between viable and dead organisms; many would be dead but capable nevertheless of being a source of biologically active substances.

The Bacteria in Cotton Dust

Dust was difficult to wet especially when the cotton had been oiled in the mills. To make an even suspension 1 g. of dust was placed in a test tube and thoroughly mixed with 5 ml. of "peptone water" (1% peptone and 0·5% NaCl. in tap water) using a glass rod. When the dust appeared completely wetted a further 10 ml. of peptone water was added, the test tube closed with a rubber bung and thoroughly shaken. The whole was transferred to a glass-stoppered bottle, made up to 100 ml., and again thoroughly shaken. From this initial dilution of 1/100 w/v other dilutions were prepared as required. (Peptone water has been used as a suspending medium and a diluent in preference to saline or distilled water as it retarded the sedimentation of the suspension.)

Viable Counts.—The results of cultural studies of cotton dust are markedly affected by the techniques and culture media employed and when assessing or comparing such results it is necessary to take this into consideration.

Preliminary observations were directed to determining the conditions which favoured the highest counts while preventing the spreading growth of some species, chiefly aerobic spore-forming bacilli, which when it occurred, rendered plates useless for counting colonies.

Pour plates were made by incorporating 1 ml. of inoculum with 15 ml. of 2% nutrient agar at 45–50°C., but they were unsuitable for counting as they were rapidly overgrown by spreaders. Sandwich plates (Fry, 1932) were therefore made from pour plates by drying them in the incubator for one hour, which fixed any organisms that were on the surface of the medium. A layer of nutrient agar was then poured over the surface and dried again for an hour which prevented spreaders growing up between the glass and the medium. Duplicate plates were made and only those having 30–300 colonies when observed with a 2½ × hand lens were counted. Sandwich plates gave proportional counts with different dilutions of inoculum up to about 300 bacteria per plate. Tests showed that the optimum times for incubation were 48 hours at 37°C. and 72 hours at 22°C. Results were consistent: four samples of one dust gave counts of 550, 560, 410, and 470 million organisms per g. with plates incubated at 37°C. for 48 hours. Inclusion in the medium of chemical inhibitors, 1/10,000 sodium azide (Snyder and Lichstein, 1940), 5–6% alcohol (Floyd and Dack, 1939), and 6%

sodium chloride (Lichstein and Snyder, 1941) will prevent the spreading of *Proteus*. These substances were therefore incorporated in pour plates, and although they prevented the spreading of bacteria present in the dust they were unsatisfactory because they reduced the number of colonies detectable in 72 hours when compared with sandwich plates. This was especially noticeable at 22°C. and may have been due to delayed growth, as further incubation produced an increase in the number of colonies, especially with alcohol which is volatile.

The results obtained with different methods of inhibiting spreaders are shown in Table 2.

TABLE 2

EFFECT OF CHEMICALS FOR INHIBITING "SPREADERS" ON THE VIABLE COUNT OF AEROBIC BACTERIA

Medium	Millions per g. Cultured at	
	37°C. for 48 hours	22°C. for 72 hours
Agar only	560	970
Agar+4% alcohol	104	141
Agar+6% NaCl	146	80
Agar+1/10,000 sodium azide..	76	below 3

Sandwich plates were clearly the most satisfactory of these methods for our purpose. Further tests showed that 6% agar (Hayward and Miles, 1943) prevented spreading growth and that surface inoculations, distributed over the surface to within 1 cm. of the edge ("spread plates"), gave even higher counts than sandwich plates. Six per cent. agar was, however, not suitable for use in pour plates, as the high temperature at which the agar had to be poured to ensure mixing, reduced the count; 4% agar did not prevent spreading growth.

The counts with 6% agar spread plates were not proportional to serial dilutions; if plates had more than 100 colonies, it would seem that some of the bacteria were inhibited when the plates were thickly seeded. Plates with approximately 100 colonies were therefore chosen for counting and yielded the highest viable counts of any of the methods tried.

Results.—Viable counts of the bacteria in dust from fine, medium, and low grades of cotton are shown in Table 3. A comparison of the counts obtained with sandwich plates and spread plates is also shown. In every case the plates were prepared at the same time from the same dilutions of dust. Surface inoculation consistently gave the highest viable counts; it appears that some of the bacteria did not grow in sandwich plates under microaerophilic conditions.

The relation between counts per gramme and those per millilitre was not constant due to the varying composition of the dust. The greatest differences occurred with samples from the air-cleaning plant which were lightest (see Table 1) and had therefore the largest volume per gramme which suggests that the bacteria were associated to a greater extent with the lighter organic matter derived from the cotton plant than the heavier inorganic material.

There appeared to be broad differences in bacterial count related to the grade of cotton as determined either by sandwich or spread plates and incubated at 37°C. or 22°C. There was some area of overlapping, but fine grades had the lowest counts, and medium grades, the highest, the low grades being intermediate. The significance of this is not clear but may be related to the origin and handling of the cotton rather than to the grade of the fibre. In spread plates the viable counts ranged between 108 and 4,000 million per g.

Considering the diverse conditions under which the cotton was grown, stored, and the samples collected the results were more uniform than might have been expected.

Counts from sandwich plates incubated at 22°C. were often higher than corresponding plates incubated at 37°C.; this occurred in eight of 13 samples (Table 3). Most of the differences were within the range of experimental error and were not correlated with the grade of cotton or source of dust.

Isolation of Aerobic Bacteria.—Spread plates of 6% nutrient agar were incubated at 37°C. for 48 hours. Colonies were identified with a hand lens supplemented by stained films when necessary. The genus *Bacillus* usually predominated; the remainder of the flora was composed of several varieties of Gram-negative bacilli. Colonies of micrococci were seen in cultures from one sample of dust only, and aerobic actinomyces from three samples* Table 4 shows the total numbers and the proportion of the two main kinds of bacteria. Every colony was taken into account. Each result represents at least two plates and usually more.

Identification of a representative sample of 21 colonies of *Bacillus* was attempted; two were *B. megatherium*, one was *B. pumilus*, the others resembled *B. pumilus*, *B. coagulans* or *B. subtilis*, differing from them in some minor respects (Bergey, 1948).

Every well separated colony of Gram-negative bacilli was subcultured; their identity is next considered.

MacConkey's agar plates were inoculated by spreading as for making counts and incubated at 37°C. for 48 hours. Some colonies of *Bacillus*

* The experience of workers at the Shirley Institute is that actinomyces tend to appear after 72 hours of incubation (personal communication).

TABLE 3

VIABLE COUNT OF 13 SAMPLES OF COTTON DUST USING SANDWICH PLATES AND 6% AGAR SPREAD PLATES INCUBATED AEROBICALLY

Sample*	Sandwich Plates				6% Nutrient Agar Spread Plates Incubated at 37°C. for 48 hrs.
	Incubated at 37°C. for 48 hrs.		Incubated at 22°C. for 72 hrs.		
	Millions per g.	Millions per ml.	Millions per g.	Millions per ml.	Millions per g.
A	65	30	73	33	—
B	240	47	263	53	418
C	152	13	133	11	206
D	80	13	77	13	156
E	66·5	21	110	34	108
F	670	184	785	215	700
G	560	184	970	319	620
G†	407	134	660	217	—
H	1,750	196	—	—	4,000
K	1,860	185	2,640	262	3,420
L	670	240	480	172	—
M	500	102	660	135	700
N	187	64	192	65	296
P	195	74	188	71	350

* Samples A—E were from fine grade cotton, F—K from medium grade, and L—P from low grade.
† After storage of the dust for 16 months in a stoppered jar in the laboratory.

grew but were disregarded. Colonies of Gram-negative bacilli were counted and every well separated colony subcultured. The viable counts of Gram-negative bacilli on this medium are shown in Table 4; comparing them with viable counts on spread 6% agar plates it will be seen that the counts on MacConkey's medium are lower, with one exception, but there is no uniformity in the proportionate reduction; it varies from 10 to 90%.

The predominant genus was *Bacterium**; a few strains were *Bact. alkaligenes*; *Achromobacter* was always present. The percentage of these genera occurring on 6% agar and on MacConkey's medium for four samples of dust is shown in Table 5.

A comparison of the two media using pure cultures indicated that MacConkey's medium inhibited some strains of *Achromobacter*. Some were completely inhibited and many others were markedly affected yielding a scanty growth of thin colonies. All strains were invariably indole-negative and did not ferment lactose. They grew in Koser's citrate and were Voges-Proskauer positive. Methyl-red tests and gelatin liquefaction were variable.

The strains of *Bacterium* were similar irrespective of the dust from which they were isolated or the medium used. With hardly any exceptions they grew in Koser's citrate and were Voges-Proskauer positive. Only very few produced indole and between one-third and one-half were methyl-red-positive and this often coincided with a capacity to liquefy gelatin. Most produced acid and gas in glucose and sucrose in 24 hours. The results in lactose were extremely variable; only a few produced acid and gas in 24 hours, the majority taking between two and three days; varieties producing acid only or non-lactose-fermenters in five days were common. On the whole these organisms were atypical *Bact. aerogenes*. Whether isolated from 6% nutrient agar or MacConkey's agar they invariably grew on both media and were the most predominant Gram-negative organisms (see Table 5).

* As defined by Topley and Wilson (1946).

TABLE 4

BACTERIA ISOLATED FROM SURFACE CULTURES ON 6% AGAR AND ON MACCONKEY'S MEDIUM INCUBATED AEROBICALLY AT 37°C.

Sample*	Total No. of Colonies	Aerobic Spore-formers			Gram-negative Bacilli			
		On 6% agar			On 6% agar			On MacConkey's Medium
		No. of Colonies	Millions per g.	%	No. of Colonies	Millions per g.	%	Millions per g.
A	96	69	—	71·9	27	—	28·1	67
B	289	198	286	68·5	91	131	31·5	12
C	103	44	88	42·7	59	118	57·3	102
D	74	35	74	47·3	39	82	52·7	36
E	54	41	82	75·9	13	26	24·1	21
F	444	409	645	92·1	35	55	7·9	112
G	226†	135	370	59·7	88	241	38·9	164
H	370	354	3,828	95·7	16	172	4·3	—
K	80	53	2,266	66·25	27	1,154	33·75	134
L	287	138	—	48·1	149	—	51·9	230
M	68	31	319	45·6	37	381	54·4	252
N	47	12	75	25·5	35	221	74·5	140
P	175	65	130	37·1	110	220	62·9	144

* Samples A—E were from fine grade cotton, F—K from medium grade, and L—P from low grade.
† Contained 3 micrococci.

TABLE 5

PERCENTAGE DISTRIBUTION OF GENERA OF GRAM-NEGATIVE BACTERIA GROWING AEROBICALLY AT 37°C. ON 6% NUTRIENT AGAR AND MACCONKEY'S MEDIUM

Sample	On 6% Nutrient Agar			On MacConkey's Medium		
	Bacterium	*Achromobacter*	*Bact. alkaligenes*	*Bacterium*	*Achromobacter*	*Bact. alkaligenes*
B	67·8	32·2	0	72·7	27·3	0
G	77·8	22·2	0	93·75	6·25	0
K	94·7	5·3	0	94·7	5·3	0
L	74·1	18·5	7·4	72·72	22·74	4·54

Some strains of *Bacterium* formed mucoid colonies on MacConkey's agar but only slightly mucoid colonies on nutrient agar. This mucoid appearance was associated with large capsules and appeared to be derived from lactose, as nutrient agar containing 1% lactose also produced markedly mucoid colonies and large capsules (Duguid, 1948).

The general picture of the aerobic flora of the dust from these different grades of cotton was remarkably similar.

Isolation of Anaerobic Bacteria.—A 1% suspension of dust "H" was heated for 10 min. at 80°C., inoculated on plates of 6% nutrient agar containing 7% horse blood, and incubated anaerobically. One strain of *Cl. welchii* type A was isolated, four other strains of *Clostridium* (not *Cl. welchii*) were accidentally lost.

Pathogenicity of Bacteria in Cotton Dust.—Each of three samples of dust was injected intramuscularly into two guinea-pigs one hour after giving 0·2 ml. of 10% calcium chloride. Five animals died from infection due to *Cl. welchii* type A. *Cl. histolyticum* was isolated from one animal. Other anaerobes were also isolated which did not seem to correspond with any recognized species; one of them was possibly pathogenic. Aerobic bacteria were also isolated from the guinea-pigs; they were not primarily pathogenic, and their identity and characters suggested that they probably came from the dust.

The injection of suspensions of cotton dust into guinea-pigs without tissue damage, e.g. intravenously, did not result in death.

After subcutaneous injection one animal died 21 days later from a *Cl. welchii* infection. Some animals died within five days, probably from infection with a *Clostridium* which was seen in films and culture but not isolated or identified; the colonies were not like those of *Cl. welchii*. Aerobes similar to those found in cotton dust were recovered from the animals.

The incidence and identification of clostridia in cotton dust merits further study. There is no indication that they are concerned in the aetiology of byssinosis or infect those exposed to the dust.

Mycological Examination of Dust

Isolation of Fungi.—Czapek-Dox agar *p*H 4·2 with the addition of 3% sucrose was used for the growth and isolation of fungi, as a combination of low *p*H and a synthetic medium prevented the growth of some aerobic spore-formers which interfere; both malt agar *p*H 4·5 and Czapek-Dox agar *p*H 7·0 supported their growth.

Identification of Predominant Genera.—The fungi have been identified by their macroscopic appearance on spread plates and by microscopic examination after staining with lactophenol containing 0·05% cotton blue. The predominant genera were *Aspergillus*, especially *A. niger*, and *Penicillium*, but other genera have been found and the plates were often rapidly overgrown by *Mucor* which prevented the identification of slower growing fungi. The incidence of the predominant genera in a number of samples of dust is shown in Table 6.

Spores seen on microscopical examination were similar to those found in cultures of *Aspergillus* and *Penicillium*. The viable count would indicate spores rather than mycelia, as fragments of hyphae which could pass through a 90-mesh sieve would normally be too small to remain viable in the dust.

The amount of contamination of different grades of cotton by fungal spores did not run parallel to the number of bacteria, but probably too few samples have been tested to generalize on this point.

TABLE 6

VIABLE COUNTS OF FUNGI GROWING ON CZAPEK-DOX MEDIUM AND PERCENTAGE INCIDENCE OF *Aspergillus* AND *Penicillium*

Sample*	Millions per g.	Millions per ml.	Percentage of Total Fungi		
			Aspergillus (all varieties)	*A. niger*	*Penicillium*
A	112	50·8	55·8	42·2	1·9
B	96	18·9	—	—	—
C	31	2·6	—	—	95
D	400	61·6	100	—	—
E	60	18·6	90	90	3·3
F	18	4·9	—	—	—
G	12	3·9	42·1	21·1	21·1
H	8·5	0·95	90·7	18·6	—
K	36	3·5	44·4	27·8	27·8
L	54	19·5	92·8	92·8	3·6
M	72	14·7	75·0	41·7	4·2
N	70	23·8	71·4	60·0	8·6
P	44	16·6	82·9	65·9	4·9

* Samples A—E were from fine grade cotton, F—K from medium grade, and L—P from low grade.

Spread plates gave much higher counts than pour plates (incubation for four days at 22°C.) and were more suitable for the isolation and identification of the fungi.

Viable Counts.—Spread plates were read after incubation for two days and four days. Some colonies were small and at this stage could not be identified. Occasionally a rapidly growing species necessitated an earlier final reading before overgrowth interfered. The results with the same samples of dust as were examined bacteriologically are given in Table 6.

On the whole the fungal flora, like the bacterial flora, is similar for cotton of different grades and origins.

Summary and Conclusions

Samples of fine dust obtained from waste removed by the cleaning machinery during the processing of different grades of cotton, as well as from the atmosphere near these machines, have been examined for their physical characters and for their content of bacteria and fungi.

Dust from the air-cleaning plant was lighter than that from the cleaning machinery and its ash

content was less. There was some indication that larger numbers of bacteria were found in the lighter organic matter derived from the cotton plant than in the heavier inorganic material.

Microscopically Gram-positive cocci and bacilli, bacterial spores, fragments of mould hyphae, and mould spores were seen. Gram-negative bacteria were not easily detected though they were revealed by culture. The Gram-positive cocci were probably dead as only once were they found in culture. As it was not feasible to estimate the total number of bacteria, fungi or their spores, the ratio between viable and dead organisms could not be determined.

The technique of counting viable bacteria requires cultural conditions which prevent spreading growth while allowing the maximum number of bacteria to develop colonies. The addition to agar of 1 : 10,000 sodium azide, 5–6% alcohol or 6% sodium chloride inhibited spreaders but reduced the counts so that none of these chemicals was satisfactory; "sandwich plates" were better than any of the chemicals tried; surface inoculation on 6% agar plates was the best method.

The viable counts of aerobic bacteria at 37°C. ranged from 108 to 4,000 millions per g. The counts at 22°C., in most samples, would have been higher, to judge from the results obtained with sandwich plates.

Among the aerobic bacteria the genus *Bacillus* usually predominated; the remainder were Gram-negative bacilli, of which about 70% or more were *Bacterium* and the rest *Achromobacter* except for a few *Alkaligenes* in one sample.

A few aerobic actinomyces were found in three samples and a few colonies of micrococci in one sample.

Details of the cultural and biochemical characters of the aerobic bacteria are given. They appeared not to be pathogenic for guinea-pigs.

Of the anaerobic bacteria, *Cl. welchii* (type A) was isolated once by direct plating on blood agar and from each of three samples of dust by inoculating dust and calcium chloride into guinea-pigs. *Cl. histolyticum* was obtained from one sample. Several other clostridia were isolated which did not correspond with any recognized species. There is no indication that these bacteria are concerned in the aetiology of byssinosis or constitute a special risk to persons exposed to the dust.

Viable counts of fungi ranged from 85 to 400 millions per g. The predominant genera were *Aspergillus*, especially *A. niger*, and *Penicillium*.

On the whole the bacterial and fungal flora was similar for cotton of different grades and origins. The possible relation of dead and viable micro-organisms in dust to the aetiology of byssinosis has yet to be determined.

We gratefully acknowledge a grant from the British Cotton Industry Research Association for technical assistance and supplies, the collaboration of the staffs of the Shirley Institute and the Nuffield Dept. of Occupational Health, and the cooperation of managements and staffs of mills which supplied the samples of cotton waste.

References

Bergey, D. H. (1948). "Bergey's Manual of Determinative Bacteriology," by Breed, R. S., Murray, E. G. D., Hitchens, A. P., and others. 6th ed. London.
Caminita, B. H., Baum, W. F., Neal, P. A., and Schneiter, R. (1947). *Publ. Hlth Bull., Wash.*, No. 297.
——, Schneiter, R., Kolb, R. W., and Neal, P. A. (1943). *Publ. Hlth Rep., Wash.*, **58**, 1165.
Duguid, J. P. (1948). *J. Path. Bact.*, **60**, 265.
Floyd, T. M., and Dack, G. M. (1939). *J. infect. Dis.*, **64**, 269.
Fry, R. M. (1932). *Brit. J. exp. Path.*, **13**, 456.
Hayward, N. J., and Miles, A. A. (1943). *Lancet*, **2**, 116.
Haworth, E., and Macdonald, A. D. (1937). *J. Hyg., Camb.*, **37**, 234.
Lichstein, H. C., and Snyder, M. L. (1941). *J. Bact.*, **42**, 653.
Macdonald, A. D., and Maitland, H. B. (1934). *J. Hyg., Camb.*, **34**, 317.
——, and Prausnitz, C. (1936). *Spec. Rep. Ser. med. Res. Coun., Lond.*, No. 212, p. 26.
Maitland, H. B., Heap, H., and Macdonald, A. D. (1932). Appendix VI Report of the Departmental Committee on Dust in Card Rooms in the Cotton Industry, p. 87. H.M.S.O., Lond.
Neal, P. A., Schneiter, R., and Caminita, B. H. (1942). *J. Amer. med. Ass.*, **119**, 1074.
Prausnitz, C. (1936). *Spec. Rep. Ser. med. Res. Coun., Lond.*, No. 212.
Prindle, B. (1934a). *Textile Research*, Boston, **4**, 555.
——(1934b). *Ibid.*, **5**, 11.
Home Office (1932). Report of the Departmental Committee on Dust in Card Rooms in the Cotton Industry. H.M.S.O., Lond.
Schilling, R. S. F. (1950). *Brit. Med. Bull.*, **7**, 52.
Schneiter, R., Neal, P. A., and Caminita, B. H. (1942). *Amer. J. publ. Hlth*, **32**, 1345.
Snyder, M. L., and Lichstein, H. C. (1940). *J. infect. Dis.*, **67**, 113.
Topley and Wilson (1946) "Principles of Bacteriology and Immunity", revised by Wilson, G. S., and Miles, A. A. 3rd ed. London.

Brit. J. industr. Med., 1952, **9**, 146.

CARDIOVASCULAR DISEASE IN COTTON WORKERS

PART II: A CLINICAL STUDY WITH SPECIAL REFERENCE TO HYPERTENSION

BY

R. S. F. SCHILLING, N. GOODMAN, and J. G. O'SULLIVAN

From the Nuffield Department of Occupational Health, Manchester University

(RECEIVED FOR PUBLICATION FEBRUARY 15, 1952)

The object of this investigation was to find out if cotton workers exposed to fine dust had a higher incidence of cardiovascular and renal disease, and in particular of hypertension, than cotton workers not so exposed.

High death rates from cardiovascular, renal, and respiratory diseases among cotton workers have been recorded by the Registrar General since 1891. In a previous paper Schilling and Goodman (1951) showed that strippers and grinders, who are more heavily exposed to fine cotton dust than any other group, had the highest death rate of cotton workers for both cardiovascular-renal and respiratory diseases.

Some of the cardiovascular mortality could be explained by methods of "book-keeping". Before 1939 the Registrar General's rules for selecting one cause of death from certificates with multiple causes meant that many respiratory deaths were allocated to the cardiovascular group, but when a correction was attempted there still appeared to be a real excess mortality from cardiovascular-renal disease among the strippers and grinders.

Clinical studies of cotton workers by Collis (1909), Prausnitz (1936), and O'Sullivan and Dingwall-Fordyce (1950), and statistical investigations of their morbidity by Bradford Hill (1930) and Harvey (1939) have confirmed a high incidence of respiratory disease among strippers and grinders, but until now the only suggestion of a cardiovascular hazard is in the Registrar General's statistics.

Since 1910 methods of dust control in cotton card-rooms have steadily improved and exposure to cotton dust has been substantially reduced. Conditions today are so changed for the better that the respiratory hazard may have been considerably reduced, and the cardiovascular-renal hazard, if ever there was one, may have disappeared.

Nevertheless, because of the interest and importance of any relationship between working conditions and cardiovascular and renal disease, it was decided to investigate clinically a group of cotton workers. An attempt was also made at the same time to determine the extent of respiratory disease under present day environmental conditions. Special attention was given to the incidence of "essential" hypertension, because it has been suggested that cotton dust exposure may be a cause of its occurrence among cotton workers (*Brit. med. J.*, 1947).

Method of Investigation

The trades included in the group exposed to dust were strippers and grinders, blow-room workers, carders, and under-carders. The group not exposed to fine cotton dust comprised weavers and warehousemen living in the same towns, and where possible, working in the same mills.

The mills chosen were those spinning raw cotton of the lower grades, and were in no way a representative sample of the cotton spinning industry. At this stage of the enquiry it was more important to choose populations likely to show positive results, so mills spinning finer grades of raw cotton and waste cotton, where there is thought to be less dust, were not included.

There were two series of examinations: series A, the first to be done, in which a group of men between the ages of 35 and 65 was examined, and series B, in which a further sample between the ages of 50 and 60 was examined.

Series A

The experimental and control groups were selected as follows.

Experimental Group (Card- and Blow-room Workers).—This group comprised all men between 35 and 65 who had worked in the card- and blow-rooms for at least 10 years. One hundred and forty-five men were originally selected from the firms' books, but on more detailed examination

42 had to be rejected for a number of reasons: because they were in fact outside the age range, or had less than 10 years' exposure to cotton dust, or had substantial exposure to other dusts. One hundred and three men therefore were included in the final analysis. No man in this group refused to be examined.

Control Group (Weavers and Warehousemen).—This group originally comprised 113 men between 35 and 65, who had worked in weaving sheds or warehouses for at least 10 years. They were selected from the firms' books to form a sample matched with the experimental group for age and length of time in the cotton industry. It was originally intended to have equal experimental and control groups, but this was not possible. Twenty men selected from the firms' books were found on detailed examination not to fulfil the necessary criteria, and had to be rejected. Ninety-three men were therefore included in the final analysis; six weavers who refused to be examined were replaced by others from the same mills. There was no reason to believe that this refusal was related to the problem under investigation. (Five men were in one department, and it appeared to be a group decision to refuse. The other man was a Christian Scientist.)

Clinical and Social Records.—All the men were interviewed and examined at their place of work. Occupational and family histories were recorded in detail. Clinical histories were taken with special reference to respiratory, renal, and cardiovascular diseases. The clinical examination was divided into two stages. The first stage comprised urine analysis, the recording of certain body measurements, clinical examination of heart and chest, and the recording of a "casual" blood pressure at the beginning of the examination. The second stage was devoted entirely to blood pressure recordings, which were made by another observer. Recordings were taken with the patient lying down, as far as possible in silence, after the procedure of the examination had been explained. Systolic and diastolic pressures were taken every three minutes and continued for 10 readings, or until the pressure stabilized at 130/70, or below. Readings were taken to the nearest multiple of 5. The technique adopted in taking pressures was that recommended by the Committee appointed by the Cardiac Society of Great Britain and Ireland (1939). All pressures were recorded on the left arm. Calibration at regular intervals showed no appreciable difference between the two manometers used, which were of the Accosson type.

Control and experimental groups were initially mixed and examined in random order to reduce the possibility of an external factor, such as emotional stress due to disturbance in the examination room, or the effect of meals, influencing one group more than the other. Where both weavers and card- and blow-room workers were working in the same mill, the observer recording pressures in the second stage of the examination did not know to which group the worker belonged, but it was not possible to make this a universal condition in the examinations.

The Findings in Series A

The two groups were comparable in respect of age and length of employment in the cotton industry (see Appendix B).

Cardiovascular and Renal Disease.—The incidence of cardiovascular and renal disease, as detected by a single clinical examination in a cotton mill, was not high. In the experimental group there was one man with rheumatic heart disease and two with renal disease. In the control group there was one man with rheumatic heart disease and one with renal disease (for details see Appendix A).

Blood Pressures.—The mean systolic and diastolic pressures of the experimental and control groups are compared in Table 1. There is a statistically significant difference at the 5% level between the "casual" diastolic pressures of the experimental and control groups. The "casual" reading, however, is probably the least reliable for this type of

TABLE 1

MEAN OF SYSTOLIC AND DIASTOLIC PRESSURES OF THE EXPERIMENTAL AND CONTROL GROUPS IN SERIES A

	Experimental Group		Control Group	
	Casual†	Lowest	Casual‡	Lowest
Number of men	100	103	92	93
Mean of systolic pressures (mm.)	141·1	127·7	141·5	127·7
S.E. of mean ..	*1·9*	*1·6*	*1·9*	*1·7*
Mean of diastolic pressures (mm.)	87·6§	82·0	84·7§	80·3
S.E. of mean ..	*1·0*	*0·9*	*1·0*	*0·9*

† 3 casual pressures not taken.
‡ 1 casual pressure not taken.
§ $t = 2{\cdot}08$, $P = 0{\cdot}04$.

TABLE 2

MEAN SYSTOLIC AND DIASTOLIC PRESSURES OF THE EXPERIMENTAL AND CONTROL GROUPS BY AGES IN SERIES A (LOWEST PRESSURES)

Age	35–		40–		50–		60–64	
	Experimental	Control	Experimental	Control	Experimental	Control	Experimental	Control
Number	12	17	50	39	31	28	10	9
Mean systolic readings	119·3	123·2	124·0	125·5	135·5	130·2	132·5	138·3
S.E. of mean ..	2·9	3·6	1·8	2·5	3·6	3·6	5·9	5·5
Mean diastolic readings	80·0	80·3	80·8	78·5	84·4	81·6	83·5	83·9
S.E. of mean ..	1·5	2·3	1·1	1·1	1·8	1·6	3·7	3·1

study of blood pressures, as it may be affected by many factors, such as apprehension about the examination, and recent exercise. Diastolic pressures in some people are difficult to judge unless repeated recordings are made. Hence this finding may have little or no clinical significance. All comparisons from now on will be based on the lowest pressures recorded in the second stage of the examination.

In Table 2 the mean systolic and diastolic pressures of the two groups are compared by ages. It is only in the 50–60 age group that both the mean systolic and diastolic pressures are higher for the card- and blow-room workers than for the weavers and warehousemen, but the difference is small and not statistically significant. Card- and blow-room workers between 60 and 64 show surprisingly low systolic pressures. There is not the expected rise with age. This finding cannot be explained satisfactorily, but the numbers in this age group are small, and it may be that the men we examined were adversely selected from the point of view of investigating hypertension. The combined effects of hypertension and of the respiratory disease, which is common among card- and blow-room workers, may have forced men with relatively mild degrees of hypertension to retire from the dusty jobs.

The age distribution of strippers and grinders and weavers at the 1931 census (more recent figures are not available) supports this suggestion. It shows that the former group have proportionately fewer men over 55 than the latter (18·5% compared with 24%).

Hypertension.—Herndon (1946) and Levy, Hillman, Stroud, and White (1944), in their studies of large groups of apparently healthy men, regarded pressures which are consistently more than 150 systolic or 90 diastolic as indicative of hypertension. We adopted a more severe standard and did not regard a man as suffering from hypertension unless both his lowest systolic and diastolic pressures were at or above both the levels of 150 and 90 respectively. The incidence of hypertension is shown in Table 3 by ages. The only obvious difference between the two groups (as one would expect from previous results) occurs in the 50–59 age group, but the difference is not statistically significant. (Clinical details of men with hypertension are given in Appendix A.)

Thus a further group, Series B, comprising all men in other mills between the ages of 50 and 60 employed for more than 10 years, was examined in an attempt to get a more definite result.

TABLE 3

INCIDENCE OF HYPERTENSION IN AGE GROUPS IN THE EXPERIMENTAL AND CONTROL GROUPS IN SERIES A

Age	35–		40–		50–		60–64	
	Experimental	Control	Experimental	Control	Experimental	Control	Experimental	Control
Number	12	17	50	39	31	28	10	9
Lowest pressures at or above 150/90..	0	1	0	1	9*	3*	2	3

* ·20>P>·10.

Series B

Twenty-eight men were examined in the experimental group and 22 in the control group. A further 10 men in the experimental group and 11 in the control group were examined, but had to be discarded for the same reasons as given in Series A. No card- or blow-room workers refused examination, but six weavers refused. Five of them, who all came from the same mill, had decided as a group not to cooperate, and they were replaced by five men from another mill in the same area. The other man refused because he said he was in good health.

The Findings in Series B

For length of time in the industry and for age, the experimental and control groups were comparable (See Appendix B).

Cardiovascular and Renal Disease.—In the experimental and control groups there were respectively one man and two men with rheumatic heart disease. No man in either group was found to have renal disease. (See Appendix A.)

Blood Pressures and Hypertension.—In Table 4 the mean systolic and diastolic pressures of, and the incidence of hypertension in, the experimental and control groups are compared.

None of the differences between the pressures of the experimental and control groups is statistically significant. The experimental group shows a higher incidence of hypertension than the control group.*

Findings for Series A and B

We will consider the two series together, as they appear to be reasonably similar for blood pressures (see Tables 2 and 4). The data for the combined series are given in Table 5 and Fig. 1.

There are significant differences between the experimental and control groups for (1) the proportion of men with hypertension, and (2) the mean systolic pressure levels.

There are similar, though not significant, differences for the mean diastolic pressures of the combined series. Similar trends are found among the men in the rejected mill. None of these results conflict in any way, but as the levels of significance are low it is all the more necessary to scrutinize these findings to see if factors not directly related to occupation may be responsible for the differences found.

TABLE 4

MEAN OF SYSTOLIC AND DIASTOLIC PRESSURES AND INCIDENCE OF HYPERTENSION IN EXPERIMENTAL AND CONTROL GROUPS IN SERIES B

	Experimental Group	Control Group
Number of men (aged 50–59)	28	22
Mean of systolic pressures ..	140·0*	129·5*
S.E. of mean	*4·3*	*2·5*
Mean of diastolic pressures ..	85·5	81·6
S.E. of mean	*2·4*	*2·0*
Lowest pressures at or above 150/90	6	1

* $t = 1{\cdot}95$; $P = {\cdot}05$ for $t = 2{\cdot}01$.

Observer Error.—The higher incidence of hypertension found among the card- and blow-room workers could quite simply be due to conscious or unconscious bias on the part of the investigators. There are two possible sources of observer error. First the difference could arise through one or all observers being biased to read high for the experimental and/or low for the control group. It was originally planned to eliminate this source of error

TABLE 5

MEAN OF SYSTOLIC AND DIASTOLIC PRESSURES AND INCIDENCE OF HYPERTENSION IN EXPERIMENTAL AND CONTROL GROUPS IN BOTH SERIES

	Experimental Group	Control Group
Number of men (aged 50–59)	59	50
Mean of systolic pressures ..	137·9*	129·9*
S.E. of mean	*2·8*	*2·2*
Mean of diastolic pressures ..	84·9†	81·6†
S.E. of mean	*1·4*	*1·3*
Lowest pressures at or above 150/90	15*	4*

* $P < {\cdot}05$. † $P > {\cdot}05$.

* In another mill, not planned to be included in either series, similar differences between the experimental and control groups were also found. Men employed in this mill were examined after the field studies on Series B had been completed. These results could not be included because one stripper and grinder out of eight, and eight weavers out of 21, refused examination, and both experimental and control groups showed higher mean systolic and diastolic pressures than the men of this age group in other mills. At all other mills the men were seen first by one of the observers or the medical officer or welfare officer employed by the mill. At this mill the men had to be seen first by the local Trade Union secretary. It was he alone who explained the purpose of the investigation and persuaded the men to cooperate. This change from the usual procedure may explain both the high lapse rate and the higher pressure levels, which may have been caused by some anxiety about the investigation on the part of the men. The results of these examinations are given in Appendix C.

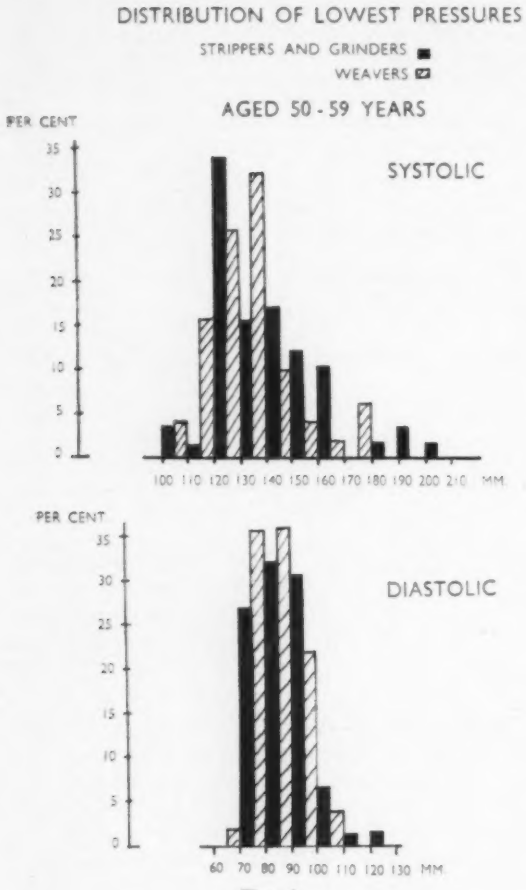


FIG. 1

by ensuring that observers who took the repeated pressures in the second stage of the examination which gave the lowest pressures would not know whether the man belonged to the experimental or control group.

This was only possible at the beginning of the investigation, when we could find mills employing both card- and blow-room workers and weavers at the same place. We can, however, test for this error by comparing the pressures of men whose jobs were known to the observer, with the pressures of men whose jobs were not known. Such a comparison is made in Table 6 for the 50–59 age group. It will be seen that there is no evidence that the knowledge of a man's job biased the observers. Secondly, another type of observer error would arise if one observer read low by comparison with other observers and took a greater proportion of the lowest pressures of the control group; or if he read high and took a greater proportion of the experimental group's pressures.

TABLE 6

THE INCIDENCE OF HYPERTENSION AND MEAN OF PRESSURE LEVELS OF MEN IN 50–59 AGE GROUP WITH JOBS UNKNOWN AND KNOWN TO THE OBSERVER IN THE COMBINED SERIES

	Experimental Group		Control Group	
	Job Known	Job Un-known	Job Known	Job Un-known
Number of men ..	46	13	22	28
Number with S/D ⩾ 150/90	11	4	1	3
Mean of systolic pressures	137·9	137·7	129·5	130·2
Mean of diastolic pressures	85·6	83·8	81·6	81·6

Six independent readings by observers on the same subjects were always within a range of 5 mm. of mercury.

For the 50–59 age group observer A took 47 of the 59 pressures of the experimental group and 40 out of 50 of the control group. As the proportion of pressures taken by the observer who took more pressures than all the other observers together was the same for both groups, it would not matter unduly if he read consistently high or low, unless he read high only for the experimental group or low only for the control group. We have already tested for the latter error in all observers and find no evidence of it, but it is possible to test specifically for this error in Observer A. The observed and expected numbers read by A, at the 150/90 level or above, for the experimental and control groups were as follows :—

50–59 AGE GROUP

	Total Number	Read by A	Total at or above 150/90 Level	At or above 150/90 Read by A	
				Ex-pected	Ob-served
Experimental group	59	47	15	11·9	12
Control group	50	40	4	4·0	3

There is no evidence that A was reading high for the experimental group or low for the control group.

Thus observer errors are not likely causes for the differences found between the pressures of the experimental and control groups. The suggestion

therefore is that the difference is a real one, and we must now examine other possible reasons for it.

Obesity and Body Measurements.—Several observations have been made on the correlation between obesity and blood-pressure, notably those by Symonds (1923) and Treadgold (1933).

In Series A the weavers as a whole were slightly heavier and shorter than the card- and blow-room workers. In series B the latter group was heavier, but the mean heights of the weavers and the card- and blow-room workers were identical. Thus it is unlikely that obesity was responsible for this difference.

Robinson and Brucer (1940) showed that body-build, as indicated by the ratio of chest circumference to height, was closely correlated with hypertension, and that obesity occurred most frequently in the lateral or broad-built types. They concluded that body-build, regardless of weight, determines in a great measure the predisposition of any individual to hypertension and that the role of obesity in hypertension is small. Among the men in the 50–59 age group, the grades of body-build described by Robinson and Brucer were similarly distributed among the experimental and control groups.

There was no evidence that the higher incidence of hypertension among the card- and blow-room workers was due to anthropometric differences between the groups.

Heredity.—Studies of family histories of patients with hypertension by Platt (1947), Weitz (1923), Hines (1937), and Ayman (1934) have shown that heredity is a factor of importance in the aetiology of "essential" hypertension. Our groups must now be examined to see if this result can be explained by heredity.

The method described by Platt (1947) for classifying family histories into four groups, positive, probable, incomplete, and negative, was adopted. In this study 42% of all the men gave incomplete family histories, whereas in Platt's series of 187 cases this proportion was only 21%. A possible explanation for this difference is that Platt's patients were ill enough to attend hospital and were probably aware of their complaint, whereas the men in our series were fit enough to work and no one of them was aware that he had hypertension. It has been stressed by Doll and Buch (1950) and others, that the patient who knows he has a particular disease is more likely to be interested in searching out similar cases in his family than the patient who is not suffering from, or does not know he has, a disease.

With such a high proportion of incomplete family histories we cannot say to what extent heredity influenced our result, although there was no evidence that it had done so. Card- and blow-room workers did not appear to have a stronger familial tendency to hypertension than weavers. Each group had 22% of men with positive family histories of hypertension.

In this series the family history did not appear to influence the blood pressure significantly. Men with positive and probably positive family histories were grouped together and compared with the men with negative family histories. An analysis of variance showed that for the systolic pressures only the variation between occupational groups was significant, but it was not so between histories for either the systolic or diastolic pressures, nor was there any evidence of a significant interaction between histories and occupational groups.

Renal Disease.—There was no difference in the incidence of renal disease found in the two groups, but the clinical methods used had to be crude, and it is possible that the higher incidence of hypertension in the card- and blow-room workers might be caused by renal disease which we failed to diagnose.

If we compare the medical histories of the two groups (all ages) for diseases such as nephritis, trench fever, and scarlet fever, the experimental and control groups respectively had 18 and 23 men with suggestive histories of renal disease. It therefore seems unlikely that hypertension secondary to undetected renal disease was the cause of the difference between the two groups.

Industrial Respiratory Disease.—The incidence and severity of industrial respiratory disease (byssinosis) among the card- and blow-room workers examined must be considered, as it may bear some relation to the incidence of hypertension.

Sixty-three per cent. of the men were found to be complaining of tightness of their chests although only 11% showed any serious disability which was most likely to be due to emphysema.

Pneumoconiosis and emphysema are important aetiological factors in pulmonary hypertension (Rosenthal, 1930; McMichael, 1948; Gelfand, 1951), but their aetiological importance in systemic hypertension or the effect of a raised pulmonary pressure on the systemic pressure has not been clearly established. Gelfand found in cases of cor pulmonale that the systemic pressure was usually normal. Parkinson and Hoyle (1937), however, found that systemic hypertension was one of the diseases most commonly associated with emphysema in a series of patients attending hospital; but these patients may have sought treatment for their emphysema because of the added complication of

INCIDENCE AND GRADE OF BYSSINOSIS AMONG THE CARD- AND BLOW-ROOM WORKERS*

Grade		Number of Men	Percentage
O	No symptoms	45	34
I	Complaining of tightness of the chest on Mondays only; symptoms not progressive	52	40
II	Above symptoms getting progressively worse and occurring on days other than Mondays. Disappearance of symptoms when not exposed to dust	16	12
III	Complaining of chest tightness on every working day and considered to have some permanent disability with effort intolerance when not at work	15	11
	Not graded because of incomplete histories	3	2
	Total	131	

* Byssinosis does not occur among weavers and warehousemen in Great Britain. Here all stages of the industrial respiratory disease of card- and blow-room workers have been included under the term byssinosis. Byssinosis, because of its definition for compensation, has often been used only for the disabling stage of the disease.

systemic hypertension which was not necessarily related to emphysema. Heimann, Castberg, Walters, David, and Meyn (1950) in a study of foundrymen in Illinois found that significantly elevated systemic blood pressure levels were related to increasing degrees of pulmonary fibrosis. In the present investigation, of the men between 50 and 60 in the experimental group, there were 17 men who had no evidence of industrial respiratory disease. Their mean pressures were 131·8 mm. systolic and 81·8 mm. diastolic and three of them had hypertension. The mean pressures of the 41 men with industrial respiratory disease were 140·0 systolic and 86·1 diastolic,* and 12 of them had hypertension. The slight differences in the mean pressures and in the incidence of hypertension are not statistically significant; the numbers are small and the reliability of the methods of diagnosing and grading byssinosis is by no means certain. Byssinosis in its earlier stages can at present only be diagnosed by the occupational and medical histories (Schilling, 1950), and it is probable that in this study there was some error in both the diagnosis and the grade. By adopting more objective methods of assessing pulmonary disability in card- and blow-room workers, it is hoped in a later investigation to get a more reliable assessment of the relationship between byssinosis and systemic hypertension, and also to test the reliability of the clinical grading of byssinosis.

* Men with Grade III byssinosis had mean pressures of 144·4/87·5 mm.
Men with Grades I and II byssinosis had mean pressures of 138·9/85·8 mm.

Discussion

The difference found in the incidence of hypertension among card- and blow-room workers and weavers is not big enough to explain the large difference in their mortality rates shown by the Registrar General for 1921–23 and 1930–32. There are explanations for this discrepancy.

It has already been shown that some of the mortality excess was due to the allocation of respiratory deaths to the cardiovascular group of diseases. The Registrar General's mortality figures relate to periods 20 and 30 years ago, when there was much more dust in the card-rooms than there is today. Although we found a substantial incidence of industrial respiratory disease in the older men, and it may well have been a hang-over from the past, it was less than the incidence 40 years ago when Collis (1909) found 90% of men of all ages affected.

As the cotton mills in which this investigation was made had no records of the men who had left, we were only able to study the population fit for work. Our data cannot, therefore, give an accurate picture of the true incidence of hypertension in the occupational groups studied. This, however, will not invalidate the relative incidence of hypertension in the two trades unless hypertension is more likely to cause unfitness and disability for work in one trade than in the other through differences in physical demands and environmental conditions. It is known that many people with benign hypertension can live a normal working life for many years without symptoms, if they are kept in ignorance of their condition and not prevented from living a normal life by their medical advisers (Naish, 1946). The physical demands of work in card- and blow-rooms and weaving sheds are more or less the same, but the combined effects on card- and blow-room workers of respiratory disease and hypertension might well make the latter disease more disabling for them than for weavers. If this does happen, it would decrease our chance of finding hypertensives among the card- and blow-room workers we examined. In this respect it may be significant that we found so few card- and blow-

room workers with hypertension in the 60–65 age group.

It is not possible to state categorically that heredity does not account for the higher incidence of hypertension among the card- and blow-room workers, because of the incompleteness of the family histories. There is, however, a suggestion that hypertension may be related to industrial pulmonary disease. This or some other occupational influence may be the cause of our findings.

If this study had been made 30 years ago when the card-rooms of British cotton mills were very dusty and if the data on cardiovascular mortality could have been analysed more accurately, the results might well have been more conclusive. It would be of interest to know if there is similar but more definite evidence of an occupational hypertension among cotton workers in the other countries where dust suppression methods in cotton mills have been even less successful. Nevertheless, in this country, in spite of the efforts to reduce dust, this investigation has shown that industrial respiratory disease still remains a serious problem among the older men employed in mills spinning the coarser grades of cotton.

Summary

This paper describes a clinical study in Lancashire cotton mills spinning and weaving the coarser grades of cotton of card- and blow-room workers, who are exposed to fine cotton dust, and weavers and warehousemen who are not so exposed. Special attention was given to the incidence of hypertension. Repeated blood pressures were taken during the examination. Men were considered to be suffering from hypertension when their lowest pressures were at or above both levels of 150 mm. of mercury for the systolic pressure and 90 mm. of mercury for the diastolic pressure.

In the age group 50–59, 15 out of 59 (25%) card- and blow-room workers had hypertension as defined, whereas four (8%) out of 50 weavers and warehousemen had hypertension. This difference is statistically significant at the 5% level.

There was no evidence that this was due to observer error, to anthropometric differences between the two groups, or was related to a higher incidence of renal disease among the card- and blow-room workers. Nor was there definite evidence to suggest that the difference was due to a greater familial tendency to essential hypertension among card- and blow-room workers than among other cotton workers.

Sixty-three per cent. of the card- and blow-room workers had symptoms of byssinosis and there was a suggestive relation between industrial respiratory disease and hypertension, which will be investigated more fully.

We should like to thank the many employers and workers whose cooperation and interest made this study possible, and in particular Mr. Henniker Heaton, of the Federation of Master Cotton Spinners' Associations Ltd., Mr. A. Roberts, of the Card and Blowing Room Operatives and Ring Spinners' Association, Mr. A. Naismith of the Amalgamated Weavers' Association; Dr. J. N. Morris of the Social Medicine Research Unit of the Medical Research Council for much advice and criticism; Professor Lane and Professor Robert Platt and other colleagues inside and outside the department for their help and advice. We would also like to thank Dr. J. Gregory and Dr. R. F. L. Logan for considerable help with the examinations; Dr. Roscoe for examining the samples of urine; and Dr. R. Murray and Mr. W. Moore of the Factory Department of the Ministry of Labour and National Service for helping us to select the cotton mills.

References

Ayman, D. (1934). *Arch. intern. Med.*, **53**, 792.
British Medical Journal (1947). **1**, 727.
Cardiac Society of Gt. Britain and Ireland (1939). *Brit. Heart J.*, **3**, 261.
Collis, E. L. (1909). Annual Report of the Chief Inspector of Factories and Workshops for 1908. Home Office [Cd. 4664]. H.M.S.O. London.
Doll, R., and Buch, J. (1950). *Ann. Eugen., Lond.*, **15**, 135.
Gelfand, M. L. (1951). *Amer. J. Med.*, **10**, 27.
Harvey, P. N. (1939). Appendix to the Report of the Departmental Committee on Compensation for Card Room Workers, p. 19. H.M.S.O., London.
Heimann, H., Castberg, H. T., Walters, F. J., David, W. D., and Meyn, A. W. (1950). U.S.P.H.S. Public Health Service Publ. No. 31, p. 55 seq.
Herndon, R. F. (1946). An Introduction to Essential Hypretension. Springfield, Illinois.
Hill, A. Bradford (1930). Ind. Health Res. Board Rep., No. 59. H.M.S.O., London.
Hines, E. A. (1937). *Ann. intern. Med.*, **11**, 593.
Levy, R. L., Hillman, C. C., Stroud, W. D., and White, P. D. (1944). *J. Amer. med. Ass.*, **126**, 829.
McMichael, J. (1948). *Edinb. med. J.*, **55**, 65.
Naish, J. M. (1946). *Bristol med.-chir. J.*, **63**, 106.
O'Sullivan, J. G., and Dingwall-Fordyce, I. (1950). The Incidence of Byssinosis in the Waste Cotton Industry. Personal communication.
Parkinson, J., and Hoyle, C. (1937). *Quart. J. Med.*, n.s. **6**, 59.
Platt, R. (1947). *Ibid.*, n.s. **16**, 111.
Prausnitz, C. (1936). *Spec. Rep. Ser. med. Res. Coun., Lond.*, No. 212.
Robinson, S. C., and Brucer, M. (1940). *Amer. J. med. Sci.*, **199**, 819.
Rosenthal, S. R. (1930). *Arch. Path., Chicago*, **10**, 717.
Schilling, R. S. F. (1950). *Brit. med. Bull.*, **7**, 52.
——, and Goodman, N. (1951). *British Journal of Industrial Medicine* **8**, 77.
Symonds, B. (1923). *J. Amer. med. Ass.*, **80**, 232.
Treadgold, H. A. (1933). *Lancet*, **1**, 733.
Weitz, W. (1923). *Z. klin. Med.*, **96**, 151.

APPENDIX A (SERIES A and B)

CASE NOTES OF MEN WITH HYPERTENSION, CARDIOVASCULAR, AND RENAL DISEASES

(A) Card- and Blow-Room Workers (Experimental Group)

Number and Series	Age	Job	Family History of Hypertension	Symptoms and Other Clinical Signs of Cardiovascular Disease	Blood Pressure		Urine	Diagnosis
					Casual or Highest	Lowest		
S27A	51	Stripper and grinder	Negative	None	160/100	150/100	No albumin	Early hypertension and byssinosis* (Grade II)
O28A	53	Stripper and grinder	Negative	None	170/95	155/95	Albumin—a trace	Early hypertension and byssinosis (Grade I)
O33A	56	Stripper and grinder	Negative	None	155/100	150/95	Albumin, occasional pus cells, and red blood cells	Early hypertension and ? chronic atrophic pyelonephritis. Byssinosis (Grade III)
S87A	55	Stripper and grinder	Probable	None	180/110	160/95	No albumin	Hypertension and byssinosis (Grade II)
O50A	56	Stripper and grinder	Probable	Apical systolic murmur	200/110	160/95	No albumin	Hypertension
S53A	59	Stripper and grinder	Positive	Retinae, nipping of veins	210/110	190/95	No albumin	Hypertension and byssinosis (Grade III)
L2A	54	Stripper and grinder	Probable	None	200/110	180/105	No albumin	Hypertension and byssinosis (Grade I)
O64A	55	Stripper and grinder	Incomplete	None	170/115	150/110	No albumin	Early hypertension and byssinosis (Grade I)
O66A	54	Stripper and grinder	Positive	None	180/105	150/95	No albumin	Early hypertension and byssinosis (Grade I)
38B	55	Stripper and grinder	Probable	Auricular fibrillation. Retinae —nipping of veins	190/110	160/90	No albumin	Hypertension and byssinosis (Grade II)
39B	53	Stripper and grinder	Probable	None	210/115	165/90	Albumin + S.G. 1010. No cells or casts seen	Hypertension and byssinosis (Grade I)
44B	57	Carder	Positive	Effort intolerance	225/125	205/115	No albumin	Hypertension and byssinosis (Grade III)
8B	57	Blow-room worker and stripper and grinder	Incomplete	None	215/115	150/95	No albumin	Hypertension
22B	57	Carder	Positive	None	190/105	160/100	No albumin	Hypertension
24B	58	Carder	Incomplete	Effort intolerance	215/140	195/125	Albumin, faint trace	Hypertension and byssinosis (Grade III)

O9A	61	Stripper and grinder	Negative	None	180/105	165/100	No albumin	Hypertension and byssinosis (Grade I)
O70A	62	Stripper and grinder	Negative	Slightly cyanosed	180/100	160/100	No albumin	Hypertension
Renal disease but no hypertension								
S19A	49	Stripper and grinder	Negative	None	145/95	135/95	Albumin, a trace. S.G. 1020. Pus cells ++	Cystitis or ? chronic atrophic pyelonephritis
Other heart disease								
L90A	35	Blow-room worker	Incomplete	Mitral systolic and diastolic murmurs	135/80	110/70	No albumin	Rheumatic heart disease
64B	55	Under-carder	Negative	Slight effort intolerance, mitral systolic and diastolic murmurs	125/85	105/75	No albumin	Rheumatic heart disease
Weavers and Warehousemen (Control Group) Series A and B								
L14A	50	Weaver	Positive	Effort intolerance	180/100	170/95	No albumin	Hypertension
S37A	52	Weaver	Negative	Retinae, nipping of veins	190/110	160/100	No albumin	Hypertension
L138A	50	Weaver	Incomplete	Apical systolic murmur and angina of effort. Effort intolerance	210/105	175/100	No albumin	Hypertension and angina pectoris
31B	58	Foreman weaver	Incomplete	None	170/105	150/95	No albumin	Early hypertension
S51A	62	Weaver	Incomplete	Frequency and scalding. Palpable right kidney	180/100	165/90	No albumin	Hypertension and ? chronic pyelonephritis
S119A	37	Weaver	Incomplete	None	165/95	150/95	No albumin	Early hypertension
L104A	60	Warehouseman	Positive	Giddiness	180/110	150/95	Albumin ++ Red blood cells and pus cells	Hypertension and chronic atrophic pyelonephritis
S143A	62	Weaver	Incomplete	None	170/100	150/90	No albumin	Early hypertension
L148A	40	Weaver	Incomplete	Tachycardia	200/95	160/90	No albumin	Hypertension
Other heart disease								
S103A	58	Weaver	Negative	Effort intolerance. Mitral systolic and diastolic murmurs	180/70	170/70	No albumin	Rheumatic heart disease
25B	52	Weaver	Negative	No symptoms. Mitral systolic and aortic diastolic murmur	160/95	135/85	No albumin	Rheumatic heart disease
49B	59	Weaver	Negative	Effort intolerance. Mitral diastolic murmur and auricular fibrillation	130/85	125/80	No albumin	Rheumatic heart disease

* For definition of byssinosis grades see page 152.

APPENDIX B

COMPARISON BETWEEN EXPERIMENTAL AND CONTROL GROUPS FOR AGE AND LENGTH OF TIME IN THE COTTON INDUSTRY

1.—Series A

Age	Experimental Group	Control Group
35—	12 } (40%)	17 } (37%)
40—	29 }	17 }
45—	21 } (33%)	22 } (41%)
50—	13 }	16 }
55—	18 } (27%)	12 } (23%)
60–64	10 }	9 }
Totals ..	103	93
Years in Industry		
10—	6 (6%)	11 (12%)
20—	36 (35%)	31 (33%)
30—	43 (42%)	38 (41%)
40—	18 (17%)	11 (12%)
50+	— —	2 (2%)
Totals ..	103	93

2.—Series B

Age	Time in Cotton Industry (Years)				
	<20	20—	30—	40—	Total
Experimental group					
50–54	—	2	12	—	14
55–59	—	1	7	6	14
Total ..	—	3	19	6	28
Control group					
50–54 ..	—	1	9	—	10
55–59 ..	2	—	3	7	12
Total ..	2	1	12	7	22

APPENDIX C

INCIDENCE OF HYPERTENSION AND MEANS OF LOWEST PRESSURES OF EXPERIMENTAL AND CONTROL GROUPS IN MILL NOT INCLUDED IN SERIES A AND B

	Experimental	Control
Number of men	7	13
No. above 150/90 level ..	3	3
Mean systolic pressure ..	152·9	135·4
S.E. of mean	*17·4*	*4·7*
Mean diastolic pressure ..	100·0	82·7
S.E. of mean	*11·7*	*3·0*

BOOK REVIEWS

Second Report of the Ministry of National Insurance for the Period 5th July, 1949, to 31st December, 1950. London: Her Majesty's Stationery Office. Pp. viii + 72. Price 2*s*. 6*d*.

This report contains much of general interest as well as matter of particular interest to industrial medicine. It gives an account of the three schemes for which the Ministry of National Insurance is responsible, family allowances, national insurance, and industrial injuries.

The number of families receiving allowances is still rising, and over 3,000,000 families now draw this allowance, at a cost of over £61 million a year. For those who fear deterioration of British family life, it may be consoling to know that in over 93% of cases the allowance was drawn by married couples living together. In only 13% of families, however, were there four or more children under 18 years of age.

The main part of the report deals with the general scheme of National Insurance. Some interesting sickness figures are given. A chart is included which gives the weekly intake of new sickness claims to June, 1951. A comparison of the last three years shows a surprisingly consistent picture except for the period of the influenza epidemic which occurred in January and February, 1951. The increased claims spread slowly from Tyneside in January, 1951, to reach the west coast a month later, and this increase later spread out from the larger centres—Manchester, Glasgow, and Dundee. The epidemic appeared to be confined to the northern half of England and Scotland.

An interesting section of the report deals with retirement. From a study carried out by sampling methods it is concluded that the higher pension (26s. a week), which began in 1946, had little or no effect on the proportion of people who continued at work after reaching pensionable age. It is, perhaps, surprising to find that among men reaching 65, 50% are still at work 18 months later, and 30% are still at work five years later, at 70. The figures for women are considerably lower. It is pointed out that at the retirement age (65 for men and 60 for women) the expectation of life is 12¾ and 18¾ years, respectively. This is a fact which must be pondered, not only by the elderly but by employed people of younger ages and by employers.

The section on industrial injuries is, perhaps, of special interest to industrial medical officers. Claims for injury benefit remain steady at 15–16,000 a week, and the coal mining industry, which represents less than 5% of the working population, accounts for a third of these claims. This would appear to be a strong argument for providing special rehabilitation facilities for this small and important section of the community. In 10% of all those claiming injury benefit the condition was sufficiently severe for them to claim disablement benefit at the end of six months. It is disappointing to see that in only 150 cases was action taken by the medical boards to see that training courses, made available by the Ministry of Labour, were used for claimants. At present three government departments are interested in the sick or injured workman, namely, the Ministries of Health, Labour and National Service, and National Insurance. The closest cooperation is obviously necessary if workers are to receive the best treatment available and the country is to benefit from their early return to work.

Special mention is made of the addition to the list of prescribed diseases of tuberculosis occurring in nurses and certain other workers in the health services. This decision introduces a new principle into "compensation", in that tuberculosis is a disease which is prevalent in the population at large. In making their recommendation to the Minister the Committee suggested that prescription of other communicable diseases should not be considered until experience had been gained from the prescription of tuberculosis.

This report is a very readable document. Of necessity there are difficulties, and certain tables relate to awkward periods. For instance, details of pneumoconiosis were not kept until April, 1950, and the report has to deal with a period of nine months. These irregularities have now been smoothed out, and reports which relate to a calendar year are promised for the future. Since nearly the whole working population is insured against sickness, the Ministry of National Insurance should give invaluable information on morbidity in this country. It is to be hoped that before long much more of this information will be widely and readily available. R. E. LANE

The Health of Welders. By A. T. Doig and L. N. Duguid. 1951. London: Her Majesty's Stationery Office. Pp. 84. Price 3*s*.

This report begins with a description of welding processes and distinguishes between the two main types, gas welding and electric welding. In both processes the welder may be exposed to fume from the metal which is being welded or from the "filler" rod which may or may not carry a coating. A mixture of iron oxides and silicates is a common type of coating for welding rods, but many other substances are already in use or are introduced as the use of alloys in industry expands. Small quantities of hydrogen, carbon dioxide, carbon monoxide, nitrous fumes, and ozone may be evolved. Both metal fume, the finely divided oxide of metal

produced by heating, and these gases may cause symptoms, especially when welding is done in confined spaces. Special risks exist when the metal which is being welded is covered with such protective coatings as zinc, which occurs on galvanized iron or steel, lead on painted metal, cadmium in paint or as an electro-plating, and the deposits from water or fuel in boilers. No information is given on the concentrations in the atmosphere of the various harmful contaminants which may occur during welding processes. These clearly depend upon the conditions of each operation and presumably on the methods of the individual workman.

The clinical section of the report begins with a critical review of the literature on health hazards of welding. In this investigation 249 men were examined from 16 firms where welding was done. Two hundred and forty-five were welders, two had previously been engaged in this trade, and two were foremen who had been exposed to the fumes in welding shops for many years. The firms were chosen so that a representative sample of welding processes and metals used would be included in the survey. Particular enquiry was made as to the type of welding upon which each man was engaged for most of the time, and each man was asked to comment upon the state of his health before he became a welder, and since taking up this occupation. He was asked also to give an opinion on the influence of the work upon his health. The average age of the group of welders examined was 34 years, the range being from 17 to 64. The average length of welding experience was 12·7 years, the longest experience being 37 years, and the shortest one year. The examination included a radiograph of the chest in 180 workmen, a controlled investigation into the effects upon the eyes in which a group of 50 shipyard welders was compared with one of 50 " shipyard non-welders " of the same age groups, and blood examinations in an unselected group of 100 welders.

The analysis of the results of the investigation is very detailed and the frequent interposition of tables and references to them in the text results in a somewhat disjointed description of the findings. These are more clearly presented in the summary.

The protection afforded by the usual clothing, gloves, and face-shield, reduces the risk of dermatitis from exposure to rays, dust, and oil, although multiple small burns result from sparks thrown off by the arc spluttering. The condition known as " arc-eyes " is common and most electric welders have experienced at least one attack. Gas welders do not suffer from this condition unless they have been exposed to unscreened rays from a neighbouring arc. The effects are temporary, but chronic conjunctivitis is found in welders more frequently than in men employed on other jobs. In the group examined no evidence of damage to the lens or deeper structures of the eye was detected. Their average age was 33·5 $\pm$ 4·9 years and their average welding experience was 10 $\pm$ 4·3 years. Cataract is seldom seen in glass workers when exposure to radiation has been for less than 17 years. No comment is made upon the nutritional state of the group of welders examined for specific lens changes. Minton (1949) states that " welders who may be suffering from some general disease, or those whose state of nutrition is poor are liable to greater damage of the lens as a result of such physical factors as infra-red radiation ".

The main positive finding was that approximately 60% of the welders examined had symptoms referable to disease of the respiratory system. Cough was the commonest complaint and abnormal physical signs were detected in about half the men who had symptoms. Of the 180 men radiographed, " definite inhalation changes " were found in 20 and " suspicious " changes in a further 12. The *x*-ray changes occur in direct proportion to the length of the employment and are more common and occur after a shorter time in electric welders. Symptoms were slightly more frequent and signs definitely more so in the group of welders showing *x*-ray changes. Two cases are described in which removal from exposure to fume resulted in retrogression of the shadows seen on *x*-ray examination. Several other welders had been found on examination 10 years previously to have *x*-ray changes which had not increased in intensity nor caused any diminution in working capacity. While this knowledge should lead to caution in assessing the significance as to prognosis of *x*-ray changes in the lungs of welders, it should not result in complacency towards the need for suppression of dust and fume especially when welding is done in confined spaces.

Doig concludes that the condition designated " welder's disease " by other observers is not a specific disease of welders due to their occupation. This, and the available evidence from statistics which suggests that there is no increased mortality or morbidity rate among welders, is reassuring. He recommends that more precise information of a statistical nature is required to ascertain that diseases such as pneumonia and gastro-intestinal disorders are no more frequent among welders than in the general population. The report concludes with a description of the regulations at present in force to protect welders from the effects of inhalation of dusts and fume, and the recommendations for the personal protection of the welder. Further recommendations designed to improve the ventilation in the vicinity of welding processes are made, and where fumes cannot be removed by exhaust ventilation the welders should be provided with breathing apparatus which ensures an adequate supply of air from an uncontaminated source. The variety of conditions which are encountered in different welding processes makes it necessary to consider each job as a separate problem if complete protection and maximum comfort is to be provided for the welder.

LESLEY BIDSTRUP

Minton, J. (1949). *Brit. med. J.*, 1, 392.

Health of Ferrous Foundrymen in Illinois. By the Division of Industrial Hygiene, U.S. Public Health Service and the Division of Industrial Hygiene, Illinois Dept. of Public Health. 1950. Public Health Service Publication No. 31. Washington, D.C.: Federal Security Agency. 24 text figures, numerous tables and diagrams. Pp. VII + 130. Price 75 cents.

It is well known that since many foundry workers are exposed to siliceous dusts they run a risk of silicosis and

considerable attention has been paid to the suppression of dust in foundries. Continuing claims for silicosis among workers in the ferrous foundry industry in three mid-western states led to a combined environmental and clinical survey.

Environmental studies were made at 18 foundries and medical studies in 16. A well illustrated account of foundry processes is first presented, followed by the environmental studies which were concentrated on the dust problems. Dust counts were made by the standard impinger apparatus. Samples for chemical analyses were collected by a vacuum cleaner on filter paper, samples being subsequently size-separated by liquid elutriation in such a way that 99% of the particles in the smaller size fraction were less than 10 microns in diameter. Free silica was determined by *x*-ray diffraction technique. The silica content of the airborne dust in the fine fraction ranged from 12 to 21%, the differences between dusts produced by different operations being relatively small. Dust concentrations were found to be considerably lower than in former investigations. The mean concentration only exceeded 10 million particles per cubic foot (approximately 300 particles/cc.) in sand-slinger moulding and in the mechanical shakeout department. Studies of other atmospheric contaminants showed low concentrations of aldehydes and low concentrations of carbon monoxide except on occasions during pouring operations in the winter months. The lighting was unsatisfactory in several plants. Sanitary facilities were, in general, good.

For purposes of correlation with the medical studies, occupational weighted dust exposures were calculated for various occupations by giving appropriate weight to the mean dust concentrations observed at the various tasks carried out in each occupation, according to the time spent in each separate task.

The medical examinations were carried out on a voluntary basis, but over 90% of the employees were examined. They consisted of a full clinical and industrial history and detailed dental examination, blood studies, a vital capacity estimation, and a full size *x*-ray film.

Details of the age distribution and marital status, personal habits, and hours of work of the men are given. For purposes of correlation of the occupation with the medical finding, the occupations were classified as principal occupation and present occupation. It was found that in over 80% of instances the present and principal occupations was identical. The various jobs were grouped together to form groups with similar dust exposure large enough in size to be related to the medical findings. Full details are given of the frequency of various abnormalities on clinical examination. There was no association between hypertension and occupation, but there was a positive association between hypertension and increasing degrees of pulmonary fibrosis as seen in the radiograph.

The largest part of the medical study was devoted to the relation of the *x*-ray finding to occupation and other abnormalities. Technical difficulties were experienced owing to variations in mains supply, and about 5% of the films taken were rejected as being technically unsatisfactory. Films were read independently by two doctors, and the films on which opinions differed were subjected to consultative opinion. The medical records of the cases were consulted when considered necessary for the proper interpretation of obscure cases. The classification adopted was that used previously by the U.S. Public Health Service, which is fully illustrated with figures and diagrams. The lower limit of definite abnormality was taken at "ground glass 2 stage" and at this level, 9·2% of the foundrymen had evidence of pulmonary fibrosis; 7·7% of the films showed ground glass 2 stage, and only 1·5% were nodular in type. Comparing this prevalence with similar studies in coal mines and metal mines in Utah, the incidence of early fibrosis was greater in the foundrymen but there was considerably less nodular fibrosis. The possibility that iron deposition in the lung may have been responsible for the radiographic abnormalities is considered, but the question is left open. The authors only point out that there was no apparent difference in the radiographic appearances in the occupations with greater exposure to iron dust than those with less exposure. No case of nodular fibrosis was found in men with less than 10 years' industrial exposure. In contrast with most previous studies, no difference was found between the frequency of nodular fibrosis in the iron and steel foundrymen. In no instance was fibrosis disabling and the vital capacity was the same in those with nodular fibrosis as in the rest of the group. When the prevalence of radiographic abnormality was compared with the weighted dust exposures, it was found to increase, as would be expected, with increase in dust exposure. There was very little difference between the different occupations, except that there was a higher incidence of fibrosis among the moulders than among the cleaners and finishers. There was no association between degree of pulmonary fibrosis and anaemia or leukocytosis but there was a slight association between pulmonary fibrosis and raised sedimentation rate. The meaning of these findings is difficult to assess because no statistical tests of significance were made.

The dental status of the workers was found to be similar to that of other industrial workers previously studied, but a curious abrasion of the surfaces of the teeth was observed which predominantly affected occupations with a higher dust exposure, and was attributed to attrition by abrasive dust in the atmosphere.

A series of recommendations for reducing dust concentrations is given, particularly to improving exhaust ventilation, to good housekeeping, and to avoiding the use of high silica parting compounds.

This volume makes little contribution to knowledge of the aetiology of silicosis. It was not conducted with this end in view. It was intended to encourage industrial hygiene and to see how serious the problem of silicosis was in the foundries studied. The volume represents a sound and unpretentious piece of work, which will be of considerable practical value to those concerned with the management of the foundry industry. There are also numerous tables showing the frequency of occurrence of different levels of blood pressure, blood counts, sedimentation rates, vital capacities, and dental abnorm-

alities in what may be regarded as a reasonably healthy industrial population. C. M. FLETCHER

Psychology and the Industrial Worker. By E. G. Chambers. 1951. Cambridge University Press. Pp. 192. Price 10*s.* 6*d.*

This book is difficult to assess. If one accepts the author's declared intention to draw illustrations " wherever possible . . . from British sources ", then it is no use complaining that 96 references from a total of 119 are British. If it is necessary that two-thirds of these British references should be to reports of the Industrial Health Research Board, then one takes leave to wonder if it was altogether wise to confine attention so narrowly to British work. We often castigate our American colleagues for their apparent inability to " read British ", but at least their all-American bibliographies are evidence of widespread sponsorship of industrial psychology.

The author devotes 27 pages to " Vocational Guidance " and 12 pages to " Vocational Selection ", but to the latter should be added an 18-page chapter on " Psychological Tests ", so the apparent emphasis is probably not measurable in pages. What is evident, however, is that the author's healthy scepticism about tests sometimes tends to negativism. He is undoubtedly at his best when explaining the uses, importance, and drawbacks of statistical methods, a subject in which he rightly enjoys a high reputation. Many little gems of wisdom are to be found, and do much to excuse what may appear to some readers a lack of balanced judgment in parts of this book. For example, no reference to factor analysis appears in the fairly full " Index of Subjects ", but on p. 149 there is an incorrect statement that psychological tests " are the sole material of the factor analyst ". Apart from the fact that nothing in the book provides the lay reader with a clue to what a " factor analyst " may be, it looks as though the author either has never read the work of Burt and Cattell, or regards personality trait ratings as included in the word " test ".

It seems a pity that in a book published in 1951 there is not even a footnote to correct the statement on p. 41 that " there has yet been no attempt to validate " the War Office Selection Board procedure. Even when Mr. Chambers' preface was written in December, 1949, it was possible to check progress in this matter ; and in April, 1950, Vernon's article " The Validation of Civil Service Selection Board Procedures " appeared in *Occupational Psychology*, a journal which must obviously be high on the author's reading list. In this paper, Professor Vernon quoted very full details of the procedure and validation, with validity coefficients of 0·5 to 0·6.

The last point which the reviewer is obliged to mention (with surprise) is the author's unfortunate inability to prevent his political views from intruding in a book of this kind. In chapter IX, which opens with the altogether praiseworthy statement that " it is possible for a psychologist to make some comments of a non-political nature, and in what follows the writer hopes that he has escaped as far as possible from the bias of party politics ". Despite his awareness of the danger, Mr. Chambers in his discussion of trade unionism and nationalized industry leaves little doubt as to which party he supports. This is a pity, especially if the book is widely read by industrial workers, for they may be inclined to distrust psychology more than they do already if the author's political views are identified with the use of psychology by management.

In summary, this book provides a useful compendium, not elsewhere available, of I.H.R.B. Reports ; a great deal of statistical and methodological sense ; and a rather lop-sided estimate of industrial psychology, its achievements and possibilities. ALASTAIR HERON

The Health of Executives : Mental Stress and Breakdown. I.W.S. Pamphlets. 1951. The Industrial Welfare Society, Inc., 48, Bryanston Square, London, W.1. Price 1*s.*

This pamphlet is a discussion of the stresses and breakdowns arising in people who exercise high mental skills and responsibilities by Dr. Cecil Roberts, Deputy Treasury Medical Adviser. After a brief survey of the nature of mental skill, Dr. Roberts considers the meaning of responsibility and concludes that it is the same as answerability. He further concludes that the crux of the stress situation is the interplay between responsibility and self-esteem. This pamphlet is of particular interest to industrial medical officers for its analysis of the early signs of breakdown and the sequence of events which occurs ; only psychological breakdowns leading to anxiety, apathy, and depression are considered, and these mainly from the angle of self-imposed stresses. Dr. Roberts gives a number of useful and practical suggestions for the avoidance of stress or at least of minimizing stress when it is inevitable. This little pamphlet contains much useful, concentrated wisdom in dealing with the affairs of a busy life ; the executive does not exist who would not derive some benefit from a study of it.

L. G. NORMAN

Human Kinetics and Analysing Body Movements. By T. McClurg Anderson. 1951. London : Wm. Heinemann Ltd. 245 text figures. Price 30*s.*

There is a growing belief that students of anatomy are being assigned too much time to the study of the cadaver. The author demonstrates in this book the possibility of teaching this factual subject in a more functional manner. Even in the introductory section, which deals with the anatomy of the body structures concerned with movement, the activities of certain muscles are illustrated by sprinting, tennis service, or golf swing, and references to sport or industrial tasks are frequent throughout the entire book. Part II deals at first with the fundamental actions performed by the limbs against external forces, and the use of gravity, elastic recoil of the tissues, and momentum as aids to maximum effort. A very good chapter follows on " Kinetic Analysis " describing " lock ", " check " and " balancing " actions and their effects, usually beneficial,

on sustained muscle action. Under the somewhat misleading title "Classification of Muscular Work", prime movers, fixation muscles, and synergists are extensively subdivided. At first sight this classification appears rather involved, but some such scheme is probably necessary to cover completely such movements as flexion of the arm at all stages. In Part III these principles are applied in formidable detail to athletics and industrial tasks. The book is profusely illustrated throughout, in many cases from films of famous athletes in action.

References to other work on the subject are not supplied and this is regrettable, however much fresh ground may have been broken by the author. The distinction between static and dynamic work is implied, but this fundamental difference might have been given more prominence, and, in view of the lengthy anatomical introduction, some space might have been devoted to oxygen consumption and efficiency of muscular work.

Many medical readers may not agree with the author's views about causation of many cases of fibrositis (strain and fatigue in certain muscles), Dupuytren's contracture (sustained contractions of muscles of the fourth and fifth fingers) or osteo-arthritis of the spine in women (stiff corsets associated with fixation of the lumbar spine).

This book is nevertheless a major contribution to the dynamics of human movements and the publisher's claim that it is essential to those concerned with athletic coaching, physical culture and education, occupational therapy, physical medicine, and industrial health, is to a large extent justified. M. L. THOMSON

Bulletin of Hygiene Reprints. Published monthly by the Bureau of Hygiene and Tropical Diseases, Keppel Street, Gower Street, London, W.C.1. Price 2*s*. Yearly subscription 20*s*. (17*s*. 6*d*. without index).

The Bureau of Hygiene and Tropical Diseases collects and disseminates information on all branches of public health and preventive medicine through the *Bulletin of Hygiene*.

The Bureau's *Bulletin of Hygiene Reprints*, which is a new publication, contains those sections of the *Bulletin of Hygiene* which deal with occupational hygiene and toxicology, atmospheric pollution, occupational psychology, physiology as applied to hygiene, and hygiene of buildings. These abstracts have been printed separately so that they may be more readily available to non-medical men such as industrial engineers, chemists, works managers, welfare officers, and others in industry who are concerned with the health and efficiency of workers. The "Reprints" will also be of value to the industrial medical officer who does not already subscribe to the *Bulletin of Hygiene*.

The abstracts, all of which are written in English by those who have special knowledge of the various subjects, reach a high standard and give useful and well classified information about original papers many of which appear in foreign or other journals to which reference is not easily made. Sometimes contributors indicate at the end of the abstract the value of the original paper. This makes the abstract much more useful and is an example which might well be followed by more contributors. RICHARD SCHILLING

THE JANUARY (1952) ISSUE

The January (1952) issue contains the following papers :—

Rheumatism in Miners. Part I : Rheumatic Complaints. By J. S. Lawrence and Jean Aitken-Swan.

A Study of the Conditions under which Methanol may Exert a Toxic Hazard in Industry. By G. Leaf and L. J. Zatman.

The Determination of Small Amounts of Tetranitromethane in Air. By V. B. Vouk and O. A. Weber.

Contribution à l'Etude de la Toxicologie du Trichloréthylène. II. By René Fabre and René Truhaut.

A Clinical-pathological Report of Eight Cases of Methyl Bromide Poisoning. By J. H. Prain and G. Harvey Smith.

Vanadium Poisoning from Cleaning Oil-fired Boilers. By N. Williams.

Salt Losses of Men Working in Hot Environments. By J. S. Weiner and R. E. van Heyningen.

Penetration of Dust Particles and Sites of Dust Stores in Pneumoconiosis. By Giacomo Mottura.

The Experimental Production of Radiographic Shadows by the Inhalation of Industrial Dusts. Part II : Zircon ($ZrSiO_4$). By H. E. Harding and T. A. Lloyd Davies.

Skin Cancer of the Hand and Forearm. By C. N. D. Cruickshank and A. Gourevitch.

Simplified Eye-piece Graticule for Assessing Thermal Precipitator Dust Samples. By H. H. Watson.

A number of copies are still available and may be obtained from the Publishing Manager, British Medical Association, Tavistock Square, W.C.1, price 12s. 6d.

ABSTRACTS

(This section of the JOURNAL *is published in collaboration with the two abstracting Journals,* Abstracts of World Medicine, *and* Abstracts of World Surgery, Obstetrics, and Gynaecology, *published by he British Medical Association. The abstracts are divided into the following sections: toxicology; industrial physiology; industrial lung disease; industrial skin diseases; accidents and orthopaedic surgery; industrial ophthalmology; environment; general. Not all sections will necessarily be represented in any one issue)*

INDUSTRIAL TOXICOLOGY

Intestinal Volvulus Precipitated by Lead Poisoning. Report of Five Cases. BERGER, K. E., and LUNDBERG, E. A. (1951). *J. Amer. med. Ass.*, **147**, 13.

Intestinal volvulus is well known in certain geographical areas as a major cause of obstruction. The authors report that the highlands of Peru fall into this category, and in some 50% of cases in natives of the area the obstruction is due to volvulus. A long-redundant, mobile megasigmoid with a narrow, pedicle-like mesosigmoid is a noticeable feature, as also is an increased dietary roughage, and possibly the hypoxia of high altitudes which commonly produced disturbances of gastro-intestinal function.

In addition to these accepted factors the authors suggest that volvulus may be precipitated by plumbism, in which the gastro-intestinal manifestation of colic is due to un-co-ordinated muscular spasms of the intestinal tract. Details are given of 5 cases, 4 involving the sigmoid colon, and 1 the small bowel. One fatality occurred owing to failure to recognize early enough that both volvulus and lead poisoning were present. All the patients had worked in a lead plant for more than 8 months and showed characteristic evidence of lead absorption. Symptoms consistent with those of gastro-intestinal plumbism preceded by a matter of days the more acute symptoms and signs heralding the onset of volvulus.

The authors feel that the temporal relation between the two conditions is so clear that it is justifiable to relate them causally, and therefore conclude that intestinal volvulus may be precipitated by lead poisoning, especially in those cases where a megasigmoid or other predisposing factor exists. *A. Lloyd Potter.*

An Appraisal of a Urinary Porphyrin Test in Detection of Lead Absorption. BROOKS, A. L. (1951). *Industr. Med. and Surg.*, **20**, 390.

A semi-quantitative method for the estimation of coproporphyrin type III, in urine, has been used by the author at General Motors Research Laboratories, Michigan, as a test for industrial lead poisoning. The method is as follows: to 5 ml. of urine in a test tube is added 1 ml. of glacial acetic acid, 5 ml. of ether, and 3 drops of hydrogen peroxide. The tube is inverted several times, and allowed to stand for at least 10 minutes. The ether layer is then read in a dark room, under ultra-violet light from a mercury vapour bulb with Wood's filter. A pale blue ether layer, devoid of pink or rose tint is read as 0, and a deep rose red is designated 4+. Lesser degrees of violet, through pink and light rose are read as 1+, 2+, and 3+, respectively.

The urines of 75 men with no history of lead exposure were all negative. In 135 urine specimens, obtained from 46 men engaged in tinning and soldering operations, in conditions where air analysis revealed no lead fumes, 105 tests were negative, but the remaining samples varied between 1 and 3+. In 742 specimens, from 128 men concerned with filing and grinding techniques, 338 showed as negative, 196 as 1+, 106 as 2+, 66 as 3+, and 36 as 4+. A positive trend to correlation between the results of urine porphyrin tests and the lead concentration in blood was observed in 100 subjects, continuously exposed to lead dust or fumes over a long period of time.

The authors consider that this simple examination of the urine is a useful supplement to other complicated tests. It is by no means a conclusive test; its main importance is that repeated negative findings are a reliable assurance that lead poisoning is not impending. *M. J. H. Smith.*

A Critical Appraisal of Current Practices in the Clinical Diagnosis of Lead Intoxication. KEHOE, R. A. (1951). *Industr. Med. and Surg.*, **20**, 253.

Poisoning by Dinitro-*ortho*-cresol. Some Observations on the Effects of Dinitro-*ortho*-cresol Administered by Mouth to Human Volunteers. HARVEY, D. G., BIDSTRUP, P. L., and BONNELL, J. A. L. (1951). *Brit. med. J.*, **2**, 13.

Five male volunteers of varying ages and weights were each given 75 mg. of pure dinitro-*ortho*-cresol (DNOC) in gelatin capsules by mouth daily for 5 or 7 days. No change in the routine of work or leisure was made in any case. The level of DNOC in the blood was estimated at frequent intervals for 12 days. For 14 days 24-hour specimens of urine were collected from each volunteer and the excretion of DNOC was determined. When administration of DNOC was completed, the effects of exercise, alcoholic and non-alcoholic drinks, and the application of DNOC to the skin were investigated. The effect of heat was not investigated. Throughout the experiments careful clinical records were kept. Graphs giving blood and urine levels of DNOC in each volunteer are included.

These experiments showed that in the human being DNOC is excreted slowly and acts as a cumulative poison. Symptoms arise after a total of 350 to 500 mg. has been given, and when the blood level is about 20 μg. per g. of blood. Exercise increases the concentration of DNOC in the blood. Similarly, it was found that small quantities of DNOC absorbed from the skin also increased the blood level. The relationship between blood concentrations of DNOC and physiological effects is discussed. *A. Thelwall Jones.*

Respiratory Complications from Tetraethyl-ammonium Ion. Report of Two Deaths. McLEMORE, G. A., GREEN, H. D., and LIDE, T. N. (1951). *Circulation*, **4**, 47.

The Toxicity of Selenium Anhydride. FILATOVA, B. C. (1951). *Gigiena*, No. 5, 18.

The formation in the atmosphere of selenium anhydride (SeO_2) may originate in the technical preparation of selenium used in the manufacture of such commodities as electrical appliances. Interest in this gas was aroused when it was discovered that it had a marked effect upon the respiratory tract and nervous system. Investigation confirmed this finding. The author carried out experiments on white rats and studied their behaviour when states of acute and chronic intoxication had been induced.

When subjected to concentrations of 0·15 to 0·6 mg. per litre (5 to 17·5 mg. per kg. body weight) the rats first showed conjunctival irritation and respiratory distress; they became restless and started rubbing their noses with their paws. After 15 to 20 minutes they began to gasp, threw back their heads, and as restlessness increased attempted to scale the walls of the chamber. The pupils dilated, convulsions set in, and the rats died in from 1 to 4 hours. Post-mortem examination revealed severe damage to the respiratory organs, indicated by oedema of the lungs. Liver, spleen, kidney, and heart showed signs of degeneration. Evidence of the effect of SeO_2 on the nervous system was given by the convulsions.

The effects of chronic intoxication were estimated from the aspect of the rats during the test, the change in weight, and the alterations in the blood picture. When subjected to a concentration of 0·01 to 0·03 mg. of SeO_2 per litre the rats lost weight, anaemia and conjunctival irritation developed, and drowsiness set in. Death occurred within 8 to 18 days. Necropsy revealed similar changes in the respiratory tract and parenchymatous organs to those present in acute intoxication. The effects of exposure to lower concentrations of the gas were much less pronounced.

Compared with other industrial poisons SeO_2 is more toxic than hydrogen phosphide and has approximately the same toxicity as phosgen. To confirm his experimental findings the author carried out an investigation in industrial premises. He found that when the concentration of the gas in the air was 0·00078 mg. per litre or greater, signs that the nervous system was affected were shown by the workers. He therefore recommends that the concentration of SeO_2 in the atmosphere should not exceed 0·0003 mg. per litre. *E. S. Fountain.*

OCCUPATIONAL CANCER

Carcinogenic Hydrocarbons and Related Compounds in Processed Rubber. FALK, H. L., STEINER, P. E., GOLDFEIN, S., BRESLOW, A., and HYKES, R. (1951). *Cancer Res.*, **11**, 318.

In experiments with rubber stoppers it was found that benzene extracts of the stoppers induced papillomata and carcinomata when painted on the skin of mice. These extracts and that from a motor-car tyre were shown to contain aromatic hydrocarbons, one of which (3 : 4-benzpyrene) is a potent carcinogen and others of which are known to be weak carcinogens. The carcinogens are most probably introduced into the rubber during the processing, in which carbon black is used. This material is made by incomplete combustion of natural gas, oils, and tars and may be the source of the carcinogens. In the processed rubber the latter are present only in small amounts and are relatively inaccessible to contact exposure. No evidence exists that exposure to products containing carbon black constitutes a practical hazard. They may be harmless under ordinary conditions of exposure, but when some organic solvents are used, as under laboratory conditions, extraction is easy and there is then danger of accidental unrecognized contamination during experiments in cancer research.

L. A. Elson.

Occupational Cancer and Other Health Hazards in a Chromate Plant: A Medical Appraisal—I. Lung Cancers in Chromate Workers. MANCUSO, T. F., and HUEPER, W. C. (1951). *Industr. Med. and Surg.*, **20**, 358.

Recent communications from the United States show, as do German figures, a high incidence of respiratory cancer in chromate workers. There are 7 principal producers of chromates in the United States, but the number of secondary industries using chromates cannot be assessed. In investigations previously reported it was shown that the working environment in a chromate plant was contaminated by trivalent and hexavalent chromium compounds, mostly the former. In the air the concentration varied from 0·06 mg. per cubic metre in offices to 1 mg. per cubic metre in certain workshops. The principal chromium compounds to which workers were exposed were the insoluble crude ore or chromite containing trivalent chromium, the soluble sodium monochromate, which is an intermediate product obtained after roasting and contains hexavalent chromium, and the soluble sodium dichromate, which is the finished product and also contains hexavalent chromium. Other raw materials used in the industry are lime, soda ash, sulphuric acid, and certain mineral sulphates. There is much dispute concerning the kind of chromate which is most harmful. Some investigators incriminate the monochromates only, having observed no cases of respiratory carcinoma in persons working exclusively with bichromates; other authors assert that free chromic acid and bichromates are not harmless; and others again are ready to absolve chromite or chrome ore, as workers mining this mineral are alleged to be free from carcinoma of the lung owing, it is supposed, to the insolubility of the chromite.

ABSTRACTS

(This section of the JOURNAL *is published in collaboration with the two abstracting Journals,* Abstracts of World Medicine, *and* Abstracts of World Surgery, Obstetrics, and Gynaecology, *published by he British Medical Association. The abstracts are divided into the following sections: toxicology; industrial physiology; industrial lung disease; industrial skin diseases; accidents and orthopaedic surgery; industrial ophthalmology; environment; general. Not all sections will necessarily be represented in any one issue)*

INDUSTRIAL TOXICOLOGY

Intestinal Volvulus Precipitated by Lead Poisoning. Report of Five Cases. BERGER, K. E., and LUNDBERG, E. A. (1951). *J. Amer. med. Ass.*, **147**, 13.

Intestinal volvulus is well known in certain geographical areas as a major cause of obstruction. The authors report that the highlands of Peru fall into this category, and in some 50% of cases in natives of the area the obstruction is due to volvulus. A long-redundant, mobile megasigmoid with a narrow, pedicle-like mesosigmoid is a noticeable feature, as also is an increased dietary roughage, and possibly the hypoxia of high altitudes which commonly produced disturbances of gastro-intestinal function.

In addition to these accepted factors the authors suggest that volvulus may be precipitated by plumbism, in which the gastro-intestinal manifestation of colic is due to un-co-ordinated muscular spasms of the intestinal tract. Details are given of 5 cases, 4 involving the sigmoid colon, and 1 the small bowel. One fatality occurred owing to failure to recognize early enough that both volvulus and lead poisoning were present. All the patients had worked in a lead plant for more than 8 months and showed characteristic evidence of lead absorption. Symptoms consistent with those of gastro-intestinal plumbism preceded by a matter of days the more acute symptoms and signs heralding the onset of volvulus.

The authors feel that the temporal relation between the two conditions is so clear that it is justifiable to relate them causally, and therefore conclude that intestinal volvulus may be precipitated by lead poisoning, especially in those cases where a megasigmoid or other predisposing factor exists. *A. Lloyd Potter.*

An Appraisal of a Urinary Porphyrin Test in Detection of Lead Absorption. BROOKS, A. L. (1951). *Industr. Med. and Surg.*, **20**, 390.

A semi-quantitative method for the estimation of coproporphyrin type III, in urine, has been used by the author at General Motors Research Laboratories, Michigan, as a test for industrial lead poisoning. The method is as follows: to 5 ml. of urine in a test tube is added 1 ml. of glacial acetic acid, 5 ml. of ether, and 3 drops of hydrogen peroxide. The tube is inverted several times, and allowed to stand for at least 10 minutes. The ether layer is then read in a dark room, under ultra-violet light from a mercury vapour bulb with Wood's filter. A pale blue ether layer, devoid of pink or rose tint is read as 0, and a deep rose red is designated 4+. Lesser degrees of violet, through pink and light rose are read as 1+, 2+, and 3+, respectively.

The urines of 75 men with no history of lead exposure were all negative. In 135 urine specimens, obtained from 46 men engaged in tinning and soldering operations, in conditions where air analysis revealed no lead fumes, 105 tests were negative, but the remaining samples varied between 1 and 3+. In 742 specimens, from 128 men concerned with filing and grinding techniques, 338 showed as negative, 196 as 1+, 106 as 2+, 66 as 3+, and 36 as 4+. A positive trend to correlation between the results of urine porphyrin tests and the lead concentration in blood was observed in 100 subjects, continuously exposed to lead dust or fumes over a long period of time.

The authors consider that this simple examination of the urine is a useful supplement to other complicated tests. It is by no means a conclusive test; its main importance is that repeated negative findings are a reliable assurance that lead poisoning is not impending. *M. J. H. Smith.*

A Critical Appraisal of Current Practices in the Clinical Diagnosis of Lead Intoxication. KEHOE, R. A. (1951). *Industr. Med. and Surg.*, **20**, 253.

Poisoning by Dinitro-*ortho*-cresol. Some Observations on the Effects of Dinitro-*ortho*-cresol Administered by Mouth to Human Volunteers. HARVEY, D. G., BIDSTRUP, P. L., and BONNELL, J. A. L. (1951). *Brit. med. J.*, **2**, 13.

Five male volunteers of varying ages and weights were each given 75 mg. of pure dinitro-*ortho*-cresol (DNOC) in gelatin capsules by mouth daily for 5 or 7 days. No change in the routine of work or leisure was made in any case. The level of DNOC in the blood was estimated at frequent intervals for 12 days. For 14 days 24-hour specimens of urine were collected from each volunteer and the excretion of DNOC was determined. When administration of DNOC was completed, the effects of exercise, alcoholic and non-alcoholic drinks, and the application of DNOC to the skin were investigated. The effect of heat was not investigated. Throughout the experiments careful clinical records were kept. Graphs giving blood and urine levels of DNOC in each volunteer are included.

These experiments showed that in the human being DNOC is excreted slowly and acts as a cumulative poison. Symptoms arise after a total of 350 to 500 mg. has been given, and when the blood level is about 20 μg. per g. of blood. Exercise increases the concentration of DNOC in the blood. Similarly, it was found that small quantities of DNOC absorbed from the skin also increased the blood level. The relationship between blood concentrations of DNOC and physiological effects is discussed. *A. Thelwall Jones.*

Respiratory Complications from Tetraethyl-ammonium Ion. Report of Two Deaths. McLEMORE, G. A., GREEN, H. D., and LIDE, T. N. (1951). *Circulation*, **4**, 47.

The Toxicity of Selenium Anhydride. FILATOVA, B. C. (1951). *Gigiena*, No. 5, 18.

The formation in the atmosphere of selenium anhydride (SeO_2) may originate in the technical preparation of selenium used in the manufacture of such commodities as electrical appliances. Interest in this gas was aroused when it was discovered that it had a marked effect upon the respiratory tract and nervous system. Investigation confirmed this finding. The author carried out experiments on white rats and studied their behaviour when states of acute and chronic intoxication had been induced.

When subjected to concentrations of 0·15 to 0·6 mg. per litre (5 to 17·5 mg. per kg. body weight) the rats first showed conjunctival irritation and respiratory distress; they became restless and started rubbing their noses with their paws. After 15 to 20 minutes they began to gasp, threw back their heads, and as restlessness increased attempted to scale the walls of the chamber. The pupils dilated, convulsions set in, and the rats died in from 1 to 4 hours. Post-mortem examination revealed severe damage to the respiratory organs, indicated by oedema of the lungs. Liver, spleen, kidney, and heart showed signs of degeneration. Evidence of the effect of SeO_2 on the nervous system was given by the convulsions.

The effects of chronic intoxication were estimated from the aspect of the rats during the test, the change in weight, and the alterations in the blood picture. When subjected to a concentration of 0·01 to 0·03 mg. of SeO_2 per litre the rats lost weight, anaemia and conjunctival irritation developed, and drowsiness set in. Death occurred within 8 to 18 days. Necropsy revealed similar changes in the respiratory tract and parenchymatous organs to those present in acute intoxication. The effects of exposure to lower concentrations of the gas were much less pronounced.

Compared with other industrial poisons SeO_2 is more toxic than hydrogen phosphide and has approximately the same toxicity as phosgen. To confirm his experimental findings the author carried out an investigation in industrial premises. He found that when the concentration of the gas in the air was 0·00078 mg. per litre or greater, signs that the nervous system was affected were shown by the workers. He therefore recommends that the concentration of SeO_2 in the atmosphere should not exceed 0·0003 mg. per litre. *E. S. Fountain.*

OCCUPATIONAL CANCER

Carcinogenic Hydrocarbons and Related Compounds in Processed Rubber. FALK, H. L., STEINER, P. E., GOLDFEIN, S., BRESLOW, A., and HYKES, R. (1951). *Cancer Res.*, **11**, 318.

In experiments with rubber stoppers it was found that benzene extracts of the stoppers induced papillomata and carcinomata when painted on the skin of mice. These extracts and that from a motor-car tyre were shown to contain aromatic hydrocarbons, one of which (3 : 4-benzpyrene) is a potent carcinogen and others of which are known to be weak carcinogens. The carcinogens are most probably introduced into the rubber during the processing, in which carbon black is used. This material is made by incomplete combustion of natural gas, oils, and tars and may be the source of the carcinogens. In the processed rubber the latter are present only in small amounts and are relatively inaccessible to contact exposure. No evidence exists that exposure to products containing carbon black constitutes a practical hazard. They may be harmless under ordinary conditions of exposure, but when some organic solvents are used, as under laboratory conditions, extraction is easy and there is then danger of accidental unrecognized contamination during experiments in cancer research.

L. A. Elson.

Occupational Cancer and Other Health Hazards in a Chromate Plant: A Medical Appraisal—I. Lung Cancers in Chromate Workers. MANCUSO, T. F., and HUEPER, W. C. (1951). *Industr. Med. and Surg.*, **20**, 358.

Recent communications from the United States show, as do German figures, a high incidence of respiratory cancer in chromate workers. There are 7 principal producers of chromates in the United States, but the number of secondary industries using chromates cannot be assessed. In investigations previously reported it was shown that the working environment in a chromate plant was contaminated by trivalent and hexavalent chromium compounds, mostly the former. In the air the concentration varied from 0·06 mg. per cubic metre in offices to 1 mg. per cubic metre in certain workshops. The principal chromium compounds to which workers were exposed were the insoluble crude ore or chromite containing trivalent chromium, the soluble sodium monochromate, which is an intermediate product obtained after roasting and contains hexavalent chromium, and the soluble sodium dichromate, which is the finished product and also contains hexavalent chromium. Other raw materials used in the industry are lime, soda ash, sulphuric acid, and certain mineral sulphates. There is much dispute concerning the kind of chromate which is most harmful. Some investigators incriminate the monochromates only, having observed no cases of respiratory carcinoma in persons working exclusively with bichromates; other authors assert that free chromic acid and bichromates are not harmless; and others again are ready to absolve chromite or chrome ore, as workers mining this mineral are alleged to be free from carcinoma of the lung owing, it is supposed, to the insolubility of the chromite.

The plant investigated by the present authors has existed for only 17 years—perhaps too short a time for full assessment of the risks. But a comprehensive investigation into the health records of all past and present workers with one year's service or more showed that out of a total of 33 deaths, 9 (27·2%) were due to cancer compared with 10·2% of all deaths in the local male population as a whole over a 12-year period. Of these 9 cases 6 involved the respiratory system, and analysis of occupation records of these individuals showed that they had been predominantly exposed to insoluble chromium compounds. It is pointed out, however, that since only 4% of all workers in this plant were exposed predominantly to soluble compounds no conclusions may be drawn concerning the relative risks from exposure to the two types of compound. Nevertheless it does appear likely that the insoluble chromite is far from being as harmless as is sometimes supposed. It is suggested that no reliable data are available for cancer deaths in chromite miners, most of whom work in remote places, and that although the ore may have an extremely low solubility, this factor, by favouring prolonged retention of particles, may indeed promote local cancerous change. By contrast, ulcers of the skin and nose caused by soluble chromate, much of which is removed rapidly by sloughing, free secretion, and absorption, do not become malignant. Tests *in vitro* show that chromite is soluble in blood to a slight degree, and the blood of workers exposed to chromite ore contains appreciable amounts of the metal, which may persist for months after they leave the industry. It is also true, however, that in persons who do not work with chromium some traces are normally found in the blood.

Post-mortem examinations were made on 3 chromate workers and all were found to have chromite dust particles in the lungs. Two died of carcinoma of the lung and one of carcinoma of the bladder. (In view of this last case it would be appropriate to study the excretion of the metal more fully. Whereas previous German and American papers give about 15 years as the period necessary for the development of carcinoma of the lung after exposure to chromium, the latent period in the cases reported in this article averaged 10·6 years.)

G. C. Pether.

INDUSTRIAL SKIN DISEASES

The Effect of BAL in the Prevention and Treatment of Experimental Skin Lesions due to Chromium Derivatives. FARRIS, G., and SICCA, U. (1951). *Rass. Med. industr.*, **20**, 169.

For the purpose of studying the efficacy of BAL in the prevention and treatment of skin lesions due to exposure to chromium derivatives, ulcers were produced as follows : the skin was sterilized with Dakin's fluid and a small area scarified with a sterile needle ; a drop of chromic acid solution (10%) was applied to the area and the place then covered with gauze. This was repeated daily for 3 days. After about 6 days a satisfactory ulcer had developed. Two ulcers were produced on the forearm of each of 3 volunteers, and to one of each pair an ointment containing 10% BAL was applied. This treatment did not sensibly accelerate healing.

A cream containing 10% BAL was next tested. One arm of each of 5 volunteers was scarified in 3 places and each site was subjected to one of the following procedures : to (*a*) a drop of 10% chromic acid was applied ; to (*b*) a drop of 10% BAL cream followed by a drop of chromic acid, and the two mixed together ; and to (*c*) a drop of cream containing no BAL followed by a drop of chromic acid, and the two mixed. The arm was maintained in a horizontal position for 15 to 20 minutes, then dressed with sterile gauze. The excipient cream was in all cases a preparation of polyglycol. This treatment was repeated on 3 successive days. On the 7th day the responses in the 5 arms were found to be identical. At (*a*) was a typical ulcer 7 to 10 mm. in diameter ; at (*c*) a small yet typical ulcer 2 to 3 mm. in diameter ; and at (*b*) no specific lesion.

In a further experiment a barrier cream containing 15% BAL applied 3 to 4 times daily to the hands and forearms of 10 volunteers was found to produce no sign of irritation or other response in 4 to 5 days.

Their results have led the authors to propose the use of a barrier cream containing 5% BAL as a protection against chromium derivatives. *J. W. Roe.*

ENVIRONMENT

Changes in Bone Marrow Pressure during Exposure to Simulated Altitude. KALSER, M. H., IVY, H. K., PEVSNER, L., MARBARGER, J. P., and IVY, A. C. (1951). *J. Aviat. Med.*, **22**, 286 and 311.

Bone infarction and necrosis have been reported as sequelae of decompression. Believing this to be a vascular phenomenon, the authors sought evidence of altered haemodynamics in the marrow cavity during exposure to simulated high altitudes.

Carotid blood pressure and femoral bone-marrow pressure were recorded simultaneously in anaesthetized dogs adequately supplied with oxygen. A control period at ground level was followed by simulated ascent to 40,000 feet (12,000 m.), pressures being recorded every 10,000 feet (3,000 m.). The maximum altitude was maintained for periods of up to 1 hour.

No consistent variation occurred in the carotid blood pressure, but the bone marrow pressure was reduced from an average of 52/41 mm. Hg at ground level to 44/41 mm. Hg at 40,000 feet. The pulse-pressure change was directly proportional to the altitude.

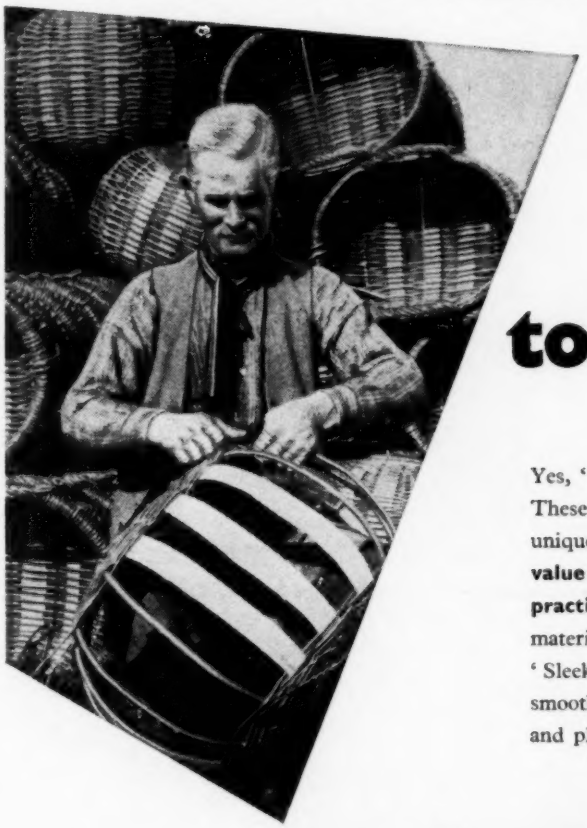
Experiments *in vitro* with an excised bone showed that under these conditions prolonged exposure to a given altitude is accompanied by a further steep rise in internal pressure. This indicates the continued release of gas within the cavity—a characteristic of dead tissues. The absence of this pressure rise in living dogs is evidence against the formation of extravascular gas bubbles.

The authors conclude that the observed fall in marrow pressure may be due either to intravascular bubble formation or to vasoconstriction, and suggest that the failure of other workers to demonstrate bubbles is in favour of the latter theory. *P. Howard.*

S.30

...ANOTHER UNUSUAL USE FOR 'Sleek'

plastic zinc oxide adhesive strapping



One of the few ancient crafts still practised all over England is basket weaving—work that is unusually hard on the fingers. Gloves are a hindrance, so many a craftsman binds a strip of 'Sleek' across vulnerable fingers—just what is needed to keep the hands free from scratches.

to emphasize

Yes, 'Sleek' does get put to some intriguing uses. These unusual jobs lend dramatic emphasis to the unique qualities which make 'Sleek' of **outstanding value for everyday use in hospital and surgery practice.** Above all, because the plastic base material is impervious to liquids, it is waterproof. 'Sleek' is washable, yet does not soil easily. It is smooth and thin yet very strong. It is extensible and pliable. It does not 'catch' or fray.

8 valuable qualities

WATERPROOF · GREASEPROOF · WASHABLE · SMOOTH
THIN · STRONG · PLIABLE · NON-FRAYING

'Sleek' TRADE MARK **plastic zinc oxide adhesive strapping**

In 2½ yd. rolls, 1" wide. In 5 yd. rolls, 1", 2", 3" and 4" wide. FREE SAMPLE sent on request.

Also

'HANDYPLAST' elastic wound dressing. With an AIR-STRIP to promote quicker healing. Strips, 1½" and 2½" wide and packs of assorted dressings.

'LEUKOPLAST' zinc oxide adhesive plaster. Laboratory controlled to ensure constant and uniform quality of the cotton cloth and adhesive strength. In 5 yd. and 10 yd. rolls—½", 1", 2" and 3" wide.

HERTS PHARMACEUTICALS LTD., WELWYN GARDEN CITY, ENGLAND

The plant investigated by the present authors has existed for only 17 years—perhaps too short a time for full assessment of the risks. But a comprehensive investigation into the health records of all past and present workers with one year's service or more showed that out of a total of 33 deaths, 9 (27·2%) were due to cancer compared with 10·2% of all deaths in the local male population as a whole over a 12-year period. Of these 9 cases 6 involved the respiratory system, and analysis of occupation records of these individuals showed that they had been predominantly exposed to insoluble chromium compounds. It is pointed out, however, that since only 4% of all workers in this plant were exposed predominantly to soluble compounds no conclusions may be drawn concerning the relative risks from exposure to the two types of compound. Nevertheless it does appear likely that the insoluble chromite is far from being as harmless as is sometimes supposed. It is suggested that no reliable data are available for cancer deaths in chromite miners, most of whom work in remote places, and that although the ore may have an extremely low solubility, this factor, by favouring prolonged retention of particles, may indeed promote local cancerous change. By contrast, ulcers of the skin and nose caused by soluble chromate, much of which is removed rapidly by sloughing, free secretion, and absorption, do not become malignant. Tests *in vitro* show that chromite is soluble in blood to a slight degree, and the blood of workers exposed to chromite ore contains appreciable amounts of the metal, which may persist for months after they leave the industry. It is also true, however, that in persons who do not work with chromium some traces are normally found in the blood.

Post-mortem examinations were made on 3 chromate workers and all were found to have chromite dust particles in the lungs. Two died of carcinoma of the lung and one of carcinoma of the bladder. (In view of this last case it would be appropriate to study the excretion of the metal more fully. Whereas previous German and American papers give about 15 years as the period necessary for the development of carcinoma of the lung after exposure to chromium, the latent period in the cases reported in this article averaged 10·6 years.)

G. C. Pether.

INDUSTRIAL SKIN DISEASES

The Effect of BAL in the Prevention and Treatment of Experimental Skin Lesions due to Chromium Derivatives. FARRIS, G., and SICCA, U. (1951). *Rass. Med. industr.*, **20,** 169.

For the purpose of studying the efficacy of BAL in the prevention and treatment of skin lesions due to exposure to chromium derivatives, ulcers were produced as follows: the skin was sterilized with Dakin's fluid and a small area scarified with a sterile needle; a drop of chromic acid solution (10%) was applied to the area and the place then covered with gauze. This was repeated daily for 3 days. After about 6 days a satisfactory ulcer had developed. Two ulcers were produced on the forearm of each of 3 volunteers, and to one of each pair an ointment containing 10% BAL was applied. This treatment did not sensibly accelerate healing.

A cream containing 10% BAL was next tested. One arm of each of 5 volunteers was scarified in 3 places and each site was subjected to one of the following procedures: to (*a*) a drop of 10% chromic acid was applied; to (*b*) a drop of 10% BAL cream followed by a drop of chromic acid, and the two mixed together; and to (*c*) a drop of cream containing no BAL followed by a drop of chromic acid, and the two mixed. The arm was maintained in a horizontal position for 15 to 20 minutes, then dressed with sterile gauze. The excipient cream was in all cases a preparation of polyglycol. This treatment was repeated on 3 successive days. On the 7th day the responses in the 5 arms were found to be identical. At (*a*) was a typical ulcer 7 to 10 mm. in diameter; at (*c*) a small yet typical ulcer 2 to 3 mm. in diameter; and at (*b*) no specific lesion.

In a further experiment a barrier cream containing 15% BAL applied 3 to 4 times daily to the hands and forearms of 10 volunteers was found to produce no sign of irritation or other response in 4 to 5 days.

Their results have led the authors to propose the use of a barrier cream containing 5% BAL as a protection against chromium derivatives. *J. W. Roe.*

ENVIRONMENT

Changes in Bone Marrow Pressure during Exposure to Simulated Altitude. KALSER, M. H., IVY, H. K., PEVSNER, L., MARBARGER, J. P., and IVY, A. C. (1951). *J. Aviat. Med.*, **22,** 286 and 311.

Bone infarction and necrosis have been reported as sequelae of decompression. Believing this to be a vascular phenomenon, the authors sought evidence of altered haemodynamics in the marrow cavity during exposure to simulated high altitudes.

Carotid blood pressure and femoral bone-marrow pressure were recorded simultaneously in anaesthetized dogs adequately supplied with oxygen. A control period at ground level was followed by simulated ascent to 40,000 feet (12,000 m.), pressures being recorded every 10,000 feet (3,000 m.). The maximum altitude was maintained for periods of up to 1 hour.

No consistent variation occurred in the carotid blood pressure, but the bone marrow pressure was reduced from an average of 52/41 mm. Hg at ground level to 44/41 mm. Hg at 40,000 feet. The pulse-pressure change was directly proportional to the altitude.

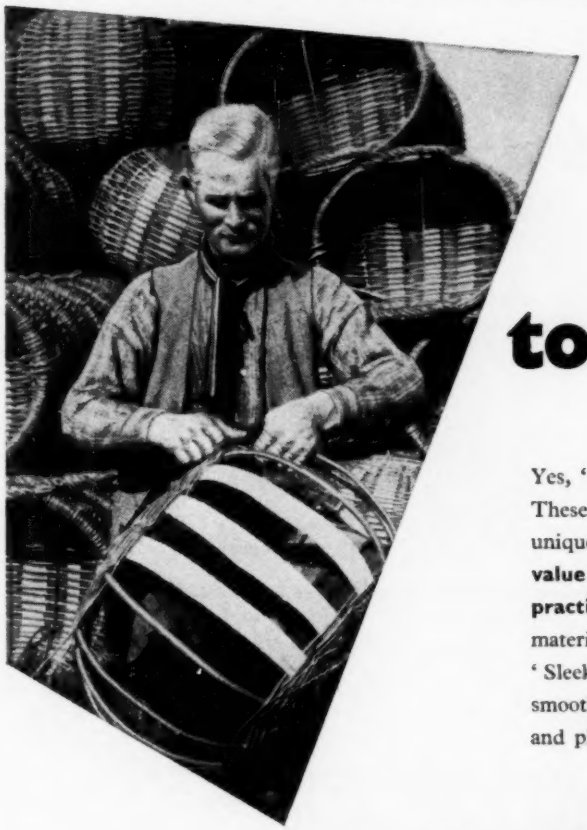
Experiments *in vitro* with an excised bone showed that under these conditions prolonged exposure to a given altitude is accompanied by a further steep rise in internal pressure. This indicates the continued release of gas within the cavity—a characteristic of dead tissues. The absence of this pressure rise in living dogs is evidence against the formation of extravascular gas bubbles.

The authors conclude that the observed fall in marrow pressure may be due either to intravascular bubble formation or to vasoconstriction, and suggest that the failure of other workers to demonstrate bubbles is in favour of the latter theory. *P. Howard.*

S.30

...ANOTHER UNUSUAL USE FOR 'Sleek'

plastic zinc oxide adhesive strapping



One of the few ancient crafts still practised all over England is basket weaving—work that is unusually hard on the fingers. Gloves are a hindrance, so many a craftsman binds a strip of 'Sleek' across vulnerable fingers—just what is needed to keep the hands free from scratches.

to emphasize

Yes, 'Sleek' does get put to some intriguing uses. These unusual jobs lend dramatic emphasis to the unique qualities which make 'Sleek' of **outstanding value for everyday use in hospital and surgery practice.** Above all, because the plastic base material is impervious to liquids, it is waterproof. 'Sleek' is washable, yet does not soil easily. It is smooth and thin yet very strong. It is extensible and pliable. It does not 'catch' or fray.

8 valuable qualities

WATERPROOF · GREASEPROOF · WASHABLE · SMOOTH
THIN · STRONG · PLIABLE · NON-FRAYING

'Sleek' plastic zinc oxide adhesive strapping

TRADE MARK

In 2½ yd. rolls, 1" wide. In 5 yd. rolls, 1", 2", 3" and 4" wide. FREE SAMPLE sent on request.

Also

'HANDYPLAST' elastic wound dressing. With an AIR-STRIP to promote quicker healing. Strips, 1½" and 2½" wide and packs of assorted dressings.

'LEUKOPLAST' zinc oxide adhesive plaster. Laboratory controlled to ensure constant and uniform quality of the cotton cloth and adhesive strength. In 5 yd. and 10 yd. rolls—½", 1", 2" and 3" wide.

HERTS PHARMACEUTICALS LTD., WELWYN GARDEN CITY, ENGLAND

RESPIRATORS *for work in Poisonous, Noxious, and Irritant Fumes*

DUST MASKS, ACIDPROOF AND WATERPROOF CLOTHING SAFETY GOGGLES, etc.

SELF-CONTAINED
OXYGEN BREATHING APPARATUS
for work in irrespirable atmospheres AND COMPRESSED AIR TYPES

RESUSCITATION APPARATUS

For reviving persons asphyxiated by poison gas or shock, Drinker Respirators (Iron Lung), OXYGEN TENTS, NASAL MASKS, GAS ANALYSIS APPARATUS, etc.

SIEBE, GORMAN & CO. LTD
LONDON, N
EVERYTHING FOR SAFETY EVERYWHERE
TOLWORTH, SURBITON, SURREY

Bandage—or barrier?

Industrial dermatitis *can* be avoided—safely, simply—if the worker uses Rozalex. It is effective against almost every known industrial irritant, and has been used in many factories for over 20 years. Quick and easy to use, Rozalex is accepted as the standard barrier preparation for industry. There is a type for every trade. May we send you free sample tins and a copy of "Skin Protection in Industry"?

ROZALEX DERMATITIS BARRIER PREPARATIONS

ROZALEX LIMITED, 10 NORFOLK STREET, MANCHESTER 2

Sternocleanse

protects the working hand

Sternol Sternocleanse

For a cost of ½d. per man per day, Sternocleanse will safeguard production and overheads. Sternocleanse is used in many of Britain's largest factories because, after severe clinical and practical tests, it proved itself to be the most satisfactory antidermatitis cream—both antiseptically and economically.

Why not make a practical test yourself? Write today for a sample tin.

COMPOSITION : *Sodium Stearate (Commercial) ; Glycerine (Pure) ; Kaolin ; plus a small percentage of Para Chlor Meta Cresol.*

STERNOL LIMITED, Royal London House, Finsbury Square, London, E.C.2
Telephone: MONarch 3871-5
Telegrams: Sternoline, Phone, London

Unexcelled for *delicate* surgery

Specially designed for the opthalmic and neuro-surgeon, the Therator Minor diathermy inspires the utmost confidence in use. Apart from a cutting current precisely suited to the most delicate surgery, absolute reliability is a positive asset. Electrical output can be finely controlled and is repeatable at pre-selected control positions. Nothing could be simpler to operate, however, as with little or no individual adjustment the Therator Minor, type MME 3, will cater for a variety of operative procedures. A *de luxe* and comprehensive set of electrodes is also available. Please write for further particulars.

The THERATOR MINOR
DIATHERMY, *Type MME 3*

MARCONI instruments

MARCONI INSTRUMENTS LTD · ST. ALBANS · HERTS · Phone: St. Albans 6161/7

Marconi House, Pudding Chare, Newcastle-on-Tyne • 233 St. Vincent Street, Glasgow • 19 The Parade, Leamington Spa
Marconi House, 38 Pall Mall, Liverpool • Marconi House, Mount Stuart Square, Cardiff • 41 Donegall Place, Belfast

CONTUSOL

Brand (Regd.)

SOLUTION OF HEPARIN

New treatment for **BRUISES, LACERATIONS** and **CONTUSED WOUNDS**

available as

CONTUSOL LOTION for general application to bruises, sprains, etc., where the skin is not broken.

CONTUSOL ANTISEPTIC with Cetrimide and Aminacrine Hydrochloride as antiseptics for treatment of contused wounds.

INDUSTRIAL MEDICAL OFFICERS
are invited to write for one or more copies of our new comprehensive Catalogue, covering Medical, Surgical and Physiotherapy requirements.

REYNOLDS & BRANSON LTD.
LEODIS WORKS
NORTH WEST ROAD, LEEDS, YORKSHIRE
Telephone : LEEDS 29351 (5 lines). Telegrams : Reynolds, Leeds

MAKERS OF THE WELL-KNOWN INDUSTRIAL HAND CLEANSER "NESCO"

DIPLOMA IN INDUSTRIAL HEALTH
of the
SOCIETY OF APOTHECARIES OF LONDON

The Society of Apothecaries of London, conscious of the paramount importance of an efficient health service in Industry, has instituted this Diploma in Industrial Health, which is of a very high standard and is open to registered medical practitioners, members of H.M. Services, and others, such as Examining Surgeons, etc.

The Examination — which consists of four Papers, Oral and Clinical — is held in July and December.

The next Examination begins on July 7th, 1952.

Regulations and Forms of Application for Admission to the Examinations may be obtained from:

The Registrar,
SOCIETY OF APOTHECARIES OF LONDON
BLACK FRIARS LANE, E.C.4

Saroul

provides safety against industrial skin diseases

Saroul Hand Cleanser has a long record of valuable service in the control and relief of industrial skin infections. It is antiseptic, emollient, beneficial to the skin — and THOROUGH ! The most ingrained grease, pitch, oil, etc., yields immediately to an application.

Full information, sample, composition, etc., on request.

SANDEMAN BROTHERS LTD
BILSLAND DRIVE
MARYHILL GLASGOW

Physiotherapy . . .

is playing an increasingly important part in the welfare departments of industrial concerns. If you are thinking of enlarging your present department or of inaugurating a new one, do not hesitate to contact us—we have been equipping the country's leading hospitals for the past 25 years. At your request, our latest illustrated catalogue will be forwarded by return of post.

ELECTRO-MEDICAL SUPPLIES

(GREENHAM) LTD.

209b GREAT PORTLAND STREET
LONDON, W.1

Opposite Met. Railway Station
Telephone : LANgham 5483-4

Telegrams : Galfaral Wesdo London
Cables : Galfaral London

MANUFACTURERS OF ELECTRO-MEDICAL AND ALLIED APPARATUS

UNIVERSITY OF MANCHESTER

NUFFIELD DEPARTMENT OF OCCUPATIONAL HEALTH

An intensive Course in Industrial Medicine will be held during the period Monday, June 23rd—Friday July 4th. The Course has been primarily designed for Assistant Industrial Medical Officers and will include lectures, symposia and demonstrations dealing with some of the basic problems of industrial medicine. Admissions to the Course will be limited to 16. Syllabus and application forms may be obtained from the Department of Occupational Health, The University, Manchester 13. All Applications should be received not later than May 23rd. The fee for the Course is £7 7s. Cheques should be made payable to The Bursar.

A PUBLICATION OF THE MEDICAL RESEARCH COUNCIL

The Application of Scientific Methods to Industrial and Service Medicine

(PROCEEDINGS OF A CONFERENCE HELD FROM MARCH 29TH TO 31ST, 1951)

3s. (3s. 3d.) [75c.]

GOVERNMENT PUBLICATIONS

Sectional List No. 12

A list of the Publications of the Medical Research Council and their Industrial Health Research Board. *Free of charge.*

Prices in brackets include postage; dollar price is post free in the United States of America.

H.M. STATIONERY OFFICE

P.O. Box 569, London, S.E.1; Edinburgh; Manchester; Birmingham; Bristol; Cardiff; Belfast; or through any bookseller; and from British Information Services, 30 Rockefeller Plaza, New York 20, U.S.A.

CONTUSOL

Brand (Regd.)

SOLUTION OF HEPARIN

New treatment for **BRUISES, LACERATIONS** and **CONTUSED WOUNDS**

available as

CONTUSOL LOTION for general application to bruises, sprains, etc., where the skin is not broken.

CONTUSOL ANTISEPTIC with Cetrimide and Aminacrine Hydrochloride as antiseptics for treatment of contused wounds.

INDUSTRIAL MEDICAL OFFICERS
are invited to write for one or more copies of our new comprehensive Catalogue, covering Medical, Surgical and Physiotherapy requirements.

REYNOLDS & BRANSON LTD.
LEODIS WORKS
NORTH WEST ROAD, LEEDS, YORKSHIRE
Telephone : LEEDS 29351 (5 lines). Telegrams : Reynolds, Leeds

MAKERS OF THE WELL-KNOWN INDUSTRIAL HAND CLEANSER "NESCO"

DIPLOMA IN INDUSTRIAL HEALTH
of the
SOCIETY OF APOTHECARIES OF LONDON

The Society of Apothecaries of London, conscious of the paramount importance of an efficient health service in Industry, has instituted this Diploma in Industrial Health, which is of a very high standard and is open to registered medical practitioners, members of H.M. Services, and others, such as Examining Surgeons, etc.

The Examination — which consists of four Papers, Oral and Clinical — is held in July and December.

The next Examination begins on July 7th, 1952.

Regulations and Forms of Application for Admission to the Examinations may be obtained from:

The Registrar,
SOCIETY OF APOTHECARIES OF LONDON
BLACK FRIARS LANE, E.C.4

Saroul

provides safety against industrial skin diseases

Saroul Hand Cleanser has a long record of valuable service in the control and relief of industrial skin infections. It is antiseptic, emollient, beneficial to the skin — and THOROUGH! The most ingrained grease, pitch, oil, etc., yields immediately to an application.

Full information, sample, composition, etc., on request.

SANDEMAN BROTHERS LTD
BILSLAND DRIVE
MARYHILL GLASGOW

EMS

Physiotherapy . . .

is playing an increasingly important part in the welfare departments of industrial concerns. If you are thinking of enlarging your present department or of inaugurating a new one, do not hesitate to contact us—we have been equipping the country's leading hospitals for the past 25 years. At your request, our latest illustrated catalogue will be forwarded by return of post.

ELECTRO-MEDICAL SUPPLIES

(GREENHAM) LTD.

209b GREAT PORTLAND STREET
LONDON, W.1

Opposite Met. Railway Station
Telephone : LANgham 3433-4

Telegrams : Galfarad Wesdo London
Cablegrams : Galfarad London

MANUFACTURERS OF ELECTRO-MEDICAL AND ALLIED APPARATUS

UNIVERSITY OF MANCHESTER

NUFFIELD DEPARTMENT OF OCCUPATIONAL HEALTH

An intensive Course in Industrial Medicine will be held during the period Monday, June 23rd—Friday July 4th. The Course has been primarily designed for Assistant Industrial Medical Officers and will include lectures, symposia and demonstrations dealing with some of the basic problems of industrial medicine. Admissions to the Course will be limited to 16. Syllabus and application forms may be obtained from the **Department of Occupational Health, The University, Manchester 13.** All Applications should be received not later than May 23rd. The fee for the Course is **£7 7s.** Cheques should be made payable to **The Bursar**.

A PUBLICATION OF THE MEDICAL RESEARCH COUNCIL

The Application of Scientific Methods to Industrial and Service Medicine

(PROCEEDINGS OF A CONFERENCE HELD FROM MARCH 29TH TO 31ST, 1951)
3*s.* (3*s.* 3*d.*) [75c.]

GOVERNMENT PUBLICATIONS

Sectional List No. 12

A list of the Publications of the Medical Research Council and their Industrial Health Research Board. *Free of charge.*

Prices in brackets include postage; dollar price is post free in the United States of America.

H. M. STATIONERY OFFICE

P.O. Box 569, London, S.E.1; Edinburgh; Manchester; Birmingham; Bristol; Cardiff; Belfast; or through any bookseller; and from British Information Services, 30 Rockefeller Plaza, New York 20, U.S.A.

13 MAY 1932

ZŌPLA

STRAPPING

Zōpla Strapping is a first-class product for surgical use. Very strong cloths, the foam is powerfully adhesive. White and Flesh Cloths, also on Elastic Cloth.

WHITE FELT

Zōpla on White Felt is very popular for padding and protective purposes. Does not harden in use.

HELVIA

First-Aid Flexible Dressing Elastic adhesive plaster with medicated gauze.

MANUFACTURED BY
LESLIES LTD
EST. 1823

HIGHAM HILL RD.
WALTHAMSTOW
LONDON, E.17.

A new approach to the treatment of eye injuries

'Acetocid' Eye Cream contains 10% (Sulphacetamide Sodium B.P.) in a stable, fluid base. The fluidity of the cream permits instillation into the eye of a drop without contamination of the cream in the tube. At body temperature, the cream liquifies immediately, spreading a soothing bacteriostatic film over the conjunctiva. 'Acetocid' Eye Cream has been specially produced for use by industrial medical officers and general practitioners in the treatment of eye injuries and infection.

'ACETOCID' EYE CREAM

Available in sealed tubes of 4 Gm. (60 grains) in containers of 1 and 36 tubes.

Samples gladly sent on request.

BS

BRITISH SCHERING LIMITED

229/231 Kensington High Street, London, W.8. telephone WEStern 8111

Printed in England by Hazell, Watson & Viney, Ltd., London and Aylesbury 2926-52